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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Annual Seminar
April 19-20-21, 1954
Kellogg Foundation Institute
Ann Arbor, Michigan

Thirty-eighth Annual Meeting
THE AMERICAN ACADEMY OF PERIODONTOLOGY
November 4, 5, 6, 1954
Miami, Florida

RAYMOND E. JOHNSON, D.D.S.

Dr. Raymond E. Johnson on January 1, 1954 turned over the duties of the Treasurer of the American Academy of Periodontology to his successor, Dr. James E. Aiguier. Dr. Johnson has been very faithful in his duties as Treasurer and while of course the new Treasurer is most capable, it is with regret that we note Dr. Johnson has decided not to be so active in the Academy. However, certainly he has done more than his share to develop the Academy to its present high standing and we wish to express publicly the Academy's sincere thanks for his many valuable contributions.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-FOUR

The Removal of Pocket and Attachment Epithelium in Humans: A Histological Study

by MELVIN L. MORRIS,* B.S., M.A., D.D.S., *New York, N. Y.*

IT has been generally held[8] that one of the prerequisites for reattachment of periodontal tissues is the removal of epithelium. The rationale presumes that the exposed connective tissue would supply the necessary elements for clot organization and repair. The method commonly used has been curetting of the epithelial wall with a sharp instrument, sometimes preceded by the application of various caustics.[7, 1, 5] The efficiency of these methods has been checked by two investigators.

Beube[3] placed sodium sulphide in certain pockets, according to McCall's technique,[7] and sodium hydroxide of identical pH in others. The tissues were removed without curettement. Only the superficial epithelial layers were chemically affected, the action being dependent on pH rather than the particular compound. Beube[2] also reported that examination of several biopsies from curetted pocket walls showed the epithelial linings to have been removed.

Waerhaug[12] curetted pocket linings in two dogs and five humans and concluded, from the histological material, that it was impossible to remove all pocket epithelium during curettage.

It was decided to study a larger human group to resolve the conflicting reports on the results of curettage.

PROCEDURE

Labial pockets on anterior teeth, destined for extraction, were chosen for ease of access and biopsy, and absence of post-operative deformity.[8] Each pocket depth was recorded and marked with a cut on the outer gingival surface (Fig. 1) as a landmark. The pocket was curetted with McCall curettes 2R and 2L, holding a finger against the external surface to immobilize it as much as possible.[4] Removal of epithelial attachment was attempted by extending the area of curettement about two millimeters beyond the pocket in all directions.

Cases No. 1 to No. 5 were run as a pilot procedure, simple curettage being performed.

*Assistant clinical professor, Division of Periodontology, School of Dental and Oral Surgery, Columbia University.

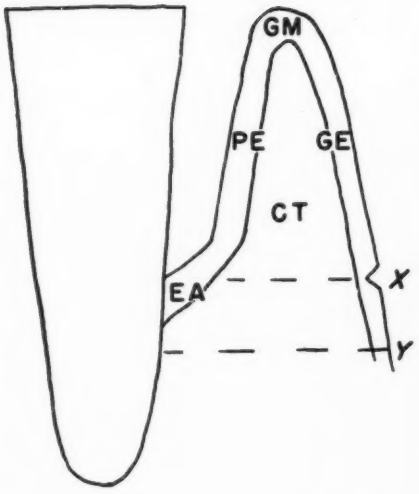

Fig. 1. GM—Gingival Margin. GE—Gingival Epithelium. PE—Pocket Epithelium. EA—Epithelium Attachment. CT—Connective Tissue. X—Landmark on outer gingiva corresponding to depth of pocket. Y—Line of biopsy incision.

Cases No. 6 to No. 15 were curetted for definitely timed intervals, the gingival marginal rim of 0.5 to 1.0 mm. cut off, and the pocket then thoroughly flushed with warm saline with a Luer syringe, 22 gauge needle.

The soft tissue components of the pockets were excised, including a margin of one to two millimeters beyond the estimated epithelial attachment (Fig. 1). The biopsies were fixed in Bouin's solution, imbedded in paraffin, and stained with hematoxylin and eosin.

RESULTS

Table I is a compilation of clinical procedures and histological results.

Cases No. 1 to No. 5, where only curettage was performed, showed a complete and consistent removal of epithelial attachment and pocket lining up to but not including the area immediately adjacent to the gingival margin. Cases No. 1 and No. 2 showed detached epithelial clumps

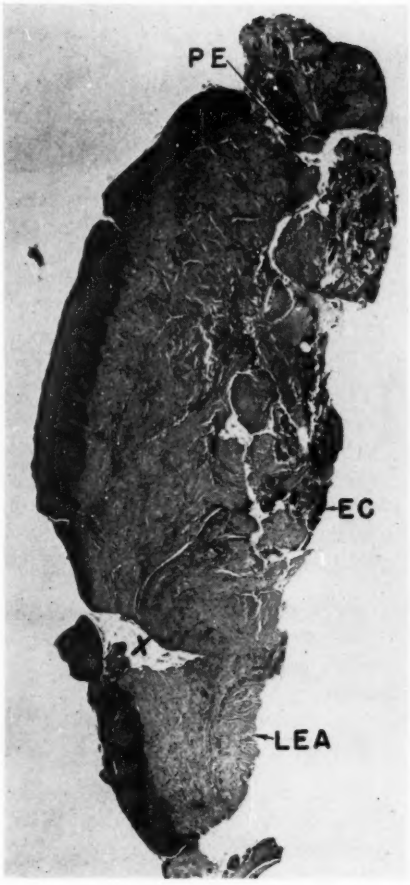

Fig. 2. EC—Epithelial Clumps. LEA—Location of former epithelial attachment. X—Landmark on outer gingiva corresponding to depth of pocket. PE—Pocket Epithelium.

in the hemorrhage. The features are seen in Figure 2, a section taken from case No. 1.

These observations suggested the procedural changes described above, namely, removal of the marginal rim and irrigation. The margin was removed because it was felt that the failure in this area was due to the great difficulty in immobilizing the tissue edge for curettage. In a further attempt at standardization, the curettage was timed. With the exception of case No. 9 (Fig. 3) where an epithelial clump was

Fig. 3. EC—Epithelial Clumps. LEA—Location of former epithelial attachment. X—Landmark on outer gingiva corresponding to depth of pocket.

Fig. 4. Epithelium completely removed. X—Landmark on outer gingiva corresponding to depth of pocket. LEA—Location of former epithelial attachment .

noted in the hemorrhage, all remaining nine cases were completely devoid of pocket and attachment epithelium. Case No. 11 (Fig. 4) is an example of this result.

Case No. 10 was unsuitable for biopsy as the curettage (2½ minutes) went completely through the soft tissue wall.

DISCUSSION

The results of this study indicate that complete epithelial removal is more easily accomplished than was indicated by Waer-

TABLE I

	PROCEDURE				LOCATION OF EPITHELIAL REMNANTS			
Case No.	Pocket Depth (mm.)	Rim Cut Off	Irrigated	Time (Min.)	Epithelial Attachment	Apical Half Pocket	Coronal Half Pocket	In Hemorrhage
1	4	–	–	–	–	–	+	+
2	4	–	–	–	–	–	+	+
3	3	–	–	–	–	–	+	–
4	5	–	–	–	–	–	+	–
5	3	–	–	–	–	–	+	–
6	3	+	+	2	–	–	–	–
7	3	+	+	2	–	–	–	–
8	4	+	+	2	–	–	–	–
9	3	+	+	2	–	–	–	+
10*	3							
11	3.5	+	+	2½	–	–	–	–
12	7	+	+	2½	–	–	–	–
13	3.5	+	+	2½	–	–	–	–
14	6	+	+	2½	–	–	–	–
15	5½	+	+	2½	–	–	–	–

* Tissue Destroyed by Curettage

haug.[12] The tactile criteria that exist in root surface curettage do not have a counterpart (for this operator) with respect to soft tissue. The author was unable, at any time, to clinically determine whether epithelium had or had not been removed. It was for this reason that timing was introduced into the experiment. Since about one quarter of the pocket was included in a curetting stroke, it may be said that curetting a soft tissue surface for approximately one half minute with a sharp curette (assuming the presence of "average skill") will be sufficient to remove epithelium. Where the tissue is thin there exists the possibility (as in Case No. 10) of completely destroying the wall.

As noted above, the epithelial attachment was uniformly and successfully removed. Epithelial attachments may vary in length from 0.25 to 6.0 mm.,[10] and from 0.4 to 1.7 mm. in a single interproximal area.[11] Further analysis of Orban and Kohler's monumental study [10] shows that although the high limit was 6.0 mm. the overwhelming majority of measurements were less than 2.0 mm. Furthermore, epithelial attachments were shortest on buccal and lingual surfaces, and where pockets were deepest. The average value obtained for labial surfaces was 0.85 mm., well within the area encompassed by the present experimental procedure.

The question arises as to the necessity or advisability of removing all epithelial attachment. Ritchey and Orban[11] deem it inadvisable because of the risk, in case of reattachment failure, of producing a pocket deeper than the original. Others[4, 6] have insisted on the necessity for complete re-

moval. It would seem to be good clinical rationale to curette an additional two millimeters to remove epithelial attachment, prompted by the theoretical possibility of growing epithelial remnants becoming a barrier to healing in the remainder of the pocket. In the possible event of deepening, the extension of two millimeters would not usually be catastrophic. This is a clinical compromise with a calculated risk, for an admittedly blind procedure.

In view of the efficacy of simple curettage in removing epithelium there is no rationale for the addition of caustics for this purpose.

SUMMARY AND CONCLUSIONS

Curettage was performed on the epithelial linings and attachments of pockets in fifteen patients. Fourteen soft tissue biopsies were obtained and examined.

Epithelial attachments and epithelial linings (up to but not including the gingival marginal area) were removed in all specimens.

In the first five cases, where only curettage was performed, epithelium always remained near the gingival margin and in two cases, were retained in the hemorrhage.

In the remaining nine cases, in addition to curettage, gingival marginal rims of 0.5 to 1.0 mm. were removed, the pockets irrigated, and the curettage timed to approximately one half minute for each area covered by a stroke of the instrument. Except for remnants in the hemorrhage in one instance, all epithelium was removed in these nine cases.

It is possible to completely remove pocket and attachment epithelium with this modified technique. The addition of caustics is felt to be completely unnecessary for this purpose.

The author wishes to express his appreciation to Dr. Barnet Levy for his advice and cooperation, to Dr. Frank E. Beube for his constructive criticisms, to Mrs. Jacqueline Brokaw for the preparation of histological material, and to Miss Elise Boyd and Drs. Stanley Heller, Bertram Klatskin, and Boas Schattan for their aid in obtaining biopsy material.

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Hereditary Gingival Fibrosis
Study of a Family*

by BHIM SEN SAVARA, B.D.S., M.S., THEODORE SUHER, D.M.D., M.S., FRANK G. EVERETT, D.M.D., M.D., ALBERT G. BURNS, D.M.D., *Portland, Oregon*

HEREDITARY gingival fibrosis (hereditary fibrous gingival hyperplasia, fibromatosis gingivae, elephantiasis gingivae) is an uncommon congenital anomaly. Literature pertaining to this condition has been reviewed recently by Hine[1], and was discussed thoroughly in the Michigan Periodontal Workshop[2].

This report deals with a family referred to the Dental School of the University of Oregon for consultation regarding excessive growth of the gingivae. Figure 1 shows the family tree. The father first showed this affliction with the eruption of his primary dentition. At fifteen years of age he had gingivectomy performed twice but the operations were not successful; as the hyperplasia recurred, he had all his teeth extracted and has been wearing full dentures since. The mother has all her own teeth and has not had any symptoms of this condition. Of the seven living children, all five girls and one of the two brothers suffer from this anomaly. The absence of the disease was noted clinically in the other brother (eight years of age in Figure 1) and confirmed later by gingival biopsy.

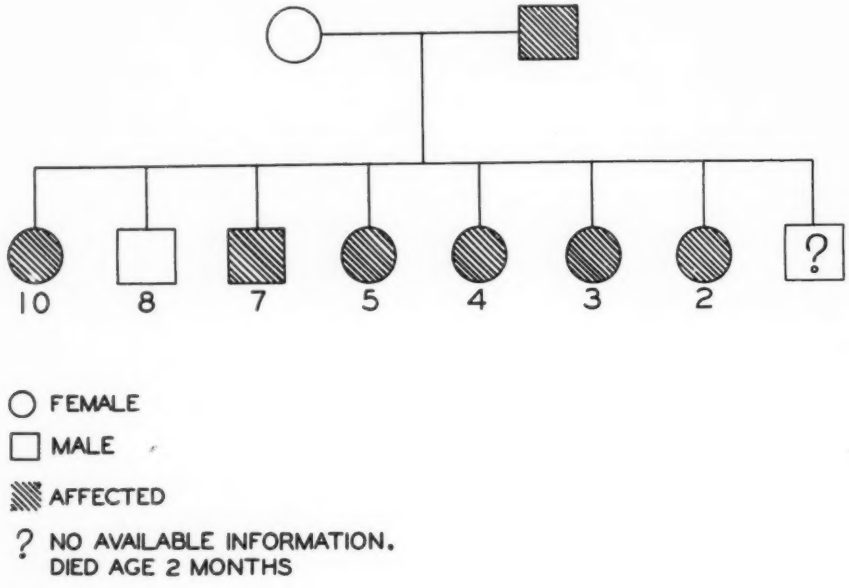

Fig. 1. Family tree. Note that only one boy is free from the anomaly.

*From the Child Study Clinic of the University of Oregon Dental School, 809 Northeast Sixth, Portland 14, Oregon. This study received financial support from the Oregon State Board of Dental Examiners.

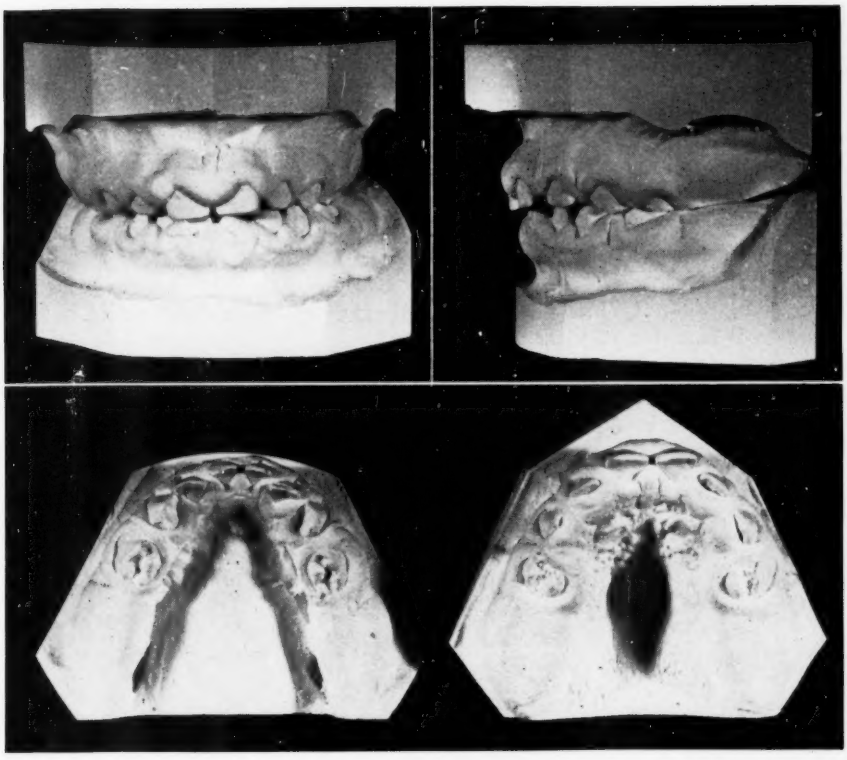

Fig. 2. Study casts of D———, three year old girl. Extensive growth of the gingival tissues can be seen in the areas of incisors and first molars. Eruption of 2nd primary molars is delayed.

One infant (youngest in the family) died at two months of age, and presence or absence of this disturbance could not be determined in him. Information regarding the gingival health of uncles, aunts, and grandparents could not be elicited in spite of repeated efforts.

CLINICAL EXAMINATION

The gingivae of the afflicted children were found to be grossly enlarged, of firm consistency, pink color and with exaggerated stippling resembling orange peel. No pain was connected with the anomaly. The condition was first observed in the children when the primary central incisors erupted. The delayed emergence of these teeth was accompanied with slight eruption gingivitis. The gingival hyperplasia increased as the children grew older and more teeth erupted.

Figure 2 illustrates the study casts of the three year old girl, D_____. The eruption of the primary second molars is delayed. The gingivae are grossly enlarged and cover nearly half the coronal portion of all the erupted teeth. Occlusion of the teeth as well as size and shape of the arches is good. Delayed eruption, as well as hyperplastic growth of the gingivae were obvious when the primary central incisors emerged at eleven months of age.

Study casts of B_____, four year old sister, are illustrated in Figure 3. The enlargement of the gingivae is advanced as compared to those of D_____ in Figure

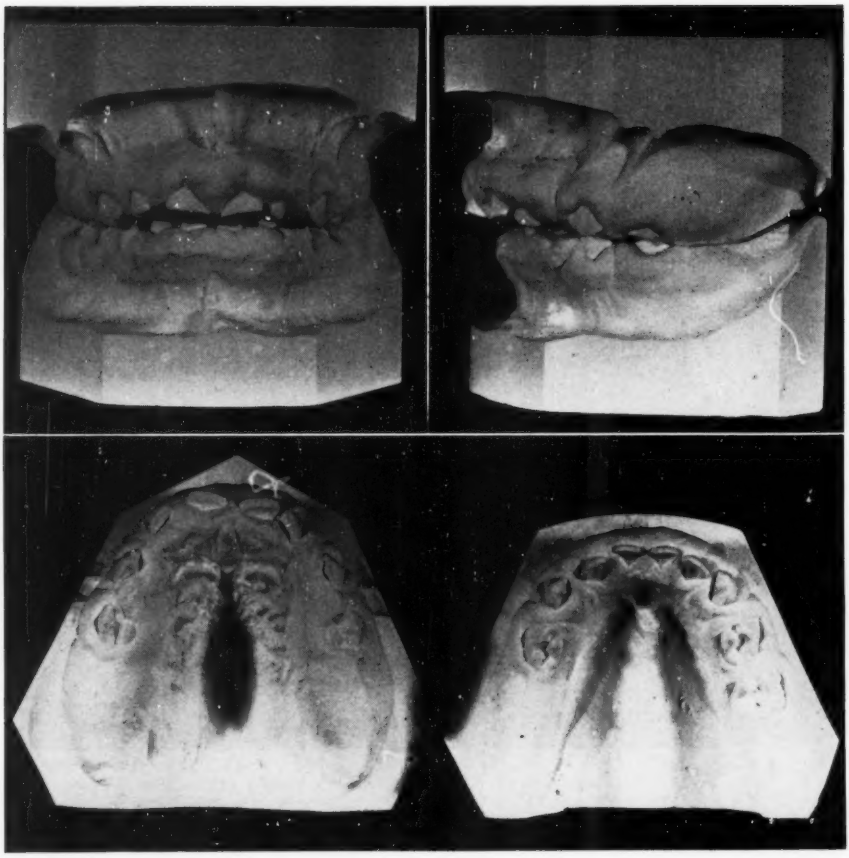

Fig. 3. Study casts of B———, four year old girl, showing extensive growth of the gingivae. Only one lower 2nd primary molar has erupted.

2. Only one primary second molar has emerged so far. A detailed comparison of the study casts of D———, B——— and K———, eldest sister (Figure 2, 3 and 4A) indicates that not only is the emergence of the teeth retarded, but the rate of eruption of the teeth after emergence in the oral cavity is decelerated. This deceleration in eruption has an adverse effect on occlusion because the growth of the jaws continues but the teeth are not reaching their level of occlusion, so that a closed bite results. This is very evident in the older affected children (Figure 3, 4A and 4B), whereas the closed bite was absent in the eight year old boy who did not have gingival hyperplasia.

Examination of intra-oral roentgenograms of the children revealed presence of all primary and permanent teeth. The resorption of the primary teeth was slow in older children. Calcification schedule of permanent teeth was within normal range. Figure 5 illustrates the roentgenograms of seven year old boy, R———, taken at five and one half years of age. The resorption of primary teeth is slow, calcification

is normal and the teeth are not ankylosed. The outline of the crypt of the calcifying bicuspid teeth, especially in the lower arch, appears rather heavy but a comparison with roentgenograms of some normal children of the same age indicated that this variation is within the range of normalcy. No other significant pathological feature was observed in the roentgenograms.

The delay in eruption could hardly be caused by any other factor but the thickness of the fibrous gingival tissue. This is well illustrated in Figure 4A, study casts of K_____ when she was eight years of age. All the primary incisors have been lost but the permanent incisors have not emerged although roentgenograms revealed that calcification of the roots was complete.

K_____'s study casts illustrate the thickened alveolar arches with deeply buried primary cuspids and first molars which are scarcely visible in the front and profile view of the occluded study casts. Permanent first molars have emerged but appear buried in the surrounding gingival

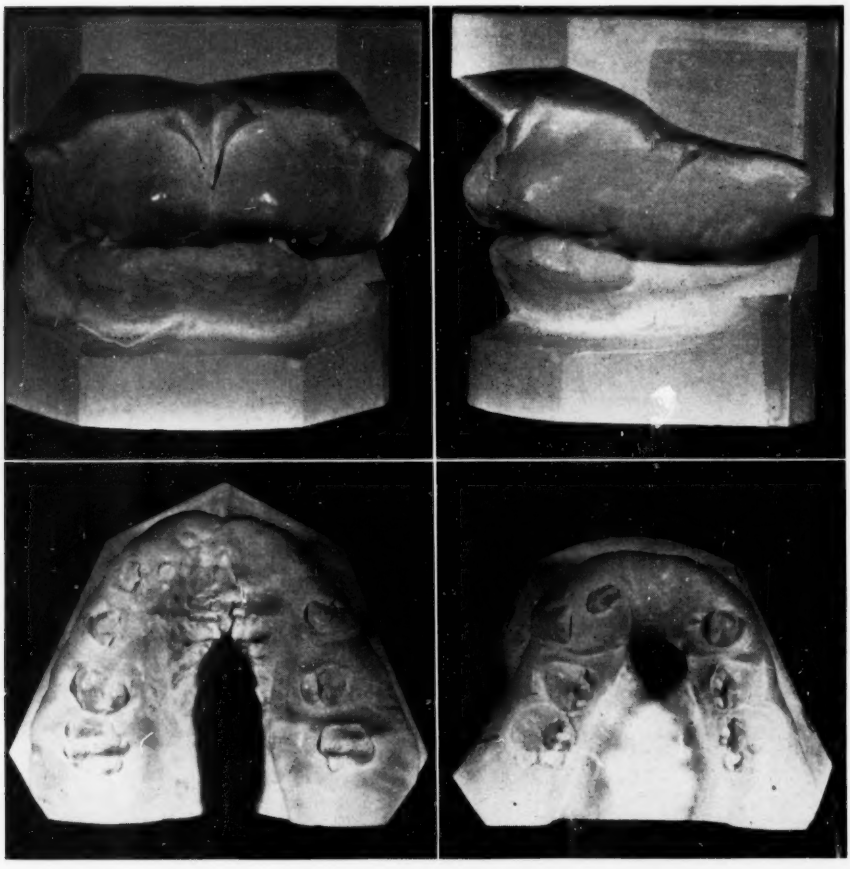

Fig. 4A. Study casts of K——— (present age ten years) taken at eight years of age before surgery. The first permanent molars have just broken through. Permanent molars and cuspids are present in the mouth. Note retardation of eruption of the permanent incisors.

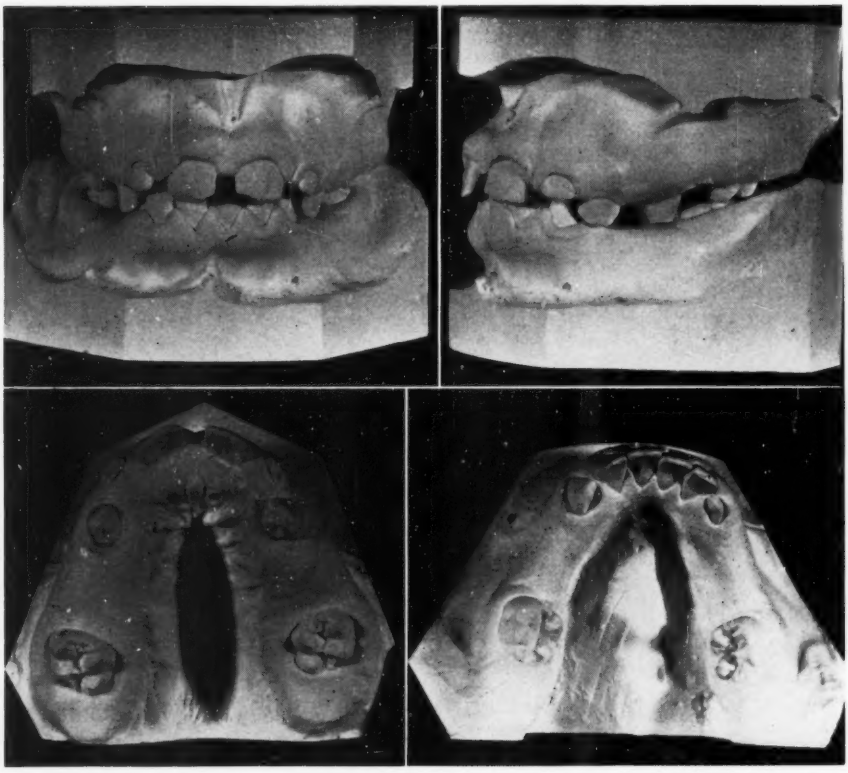

Fig. 4B. Study casts of K———, six months after surgery. Permanent incisors and first molars are fully erupted and occluding. The gingivae around the incisors appear hyperplastic.

tissue. K———— was unable to masticate properly, but did not have any apparent personality problem. The gingival growth was so excessive as to crowd the tongue. Facial profile and frontal views gave the appearance of the oral cavity being overfilled with the gingival tissues. Speech was impaired slightly. The children became conscious of the anomaly as they were examined by different specialists.

General health of the family is good. Complete physical examination of the two oldest affected children by the pediatrician did not reveal any pathology. Routine blood tests, and urinalysis yielded figures within normal ranges except for a slight eosinophilia (9 eosinophils in the peripheral blood) in the oldest child. Physique as determined by plotting height and weight on the Wetzel Grid[3] and Meredith's Chart[4] was classified as good. Skeletal maturation was determined by hand and wrist roentgenograms[5] and was found to be within the range of normalcy.

On the basis of the universal gingival hyperplasia, the hard fibrous consistency of the anomalous tissues, the relative absence of inflammatory symptoms and the apparently familiar distribution of the affliction, the condition was clinically diagnosed as hereditary gingival fibrosis.

HISTOPATHOLOGY

Gingival biopsy specimens were obtained

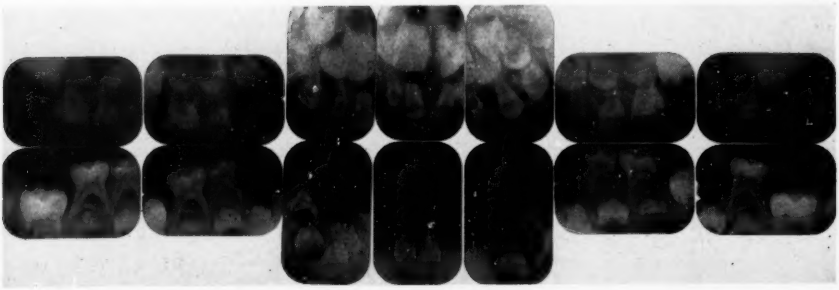

Fig. 5. Intra-oral roentgenograms of R———, seven year old boy, secured at 5½ years of age. Slow resorption of the primary teeth is seen.

from three of the children. The tissues removed in the operation of gingivectomy were also used for microscopic examination. In one non-afflicted sibling, the tissue of one gingival papilla and the adjoining marginal and attached gingiva were excised. The material was fixed in 10% formalin. The sections were cut at right angles to the long axis of the tooth. Specimens were stained with H & E, hematoxylin phloxin for hyalin[6], elasticatrichrome for elastic fibers[7], and with Wilder's silver stain.[8]

The histologic findings in the afflicted members were so similar, that a separate discussion of each individual specimen seems redundant. One single description will therefore be given.

The thickness of the epithelium appears reduced between epithelial ridges and these

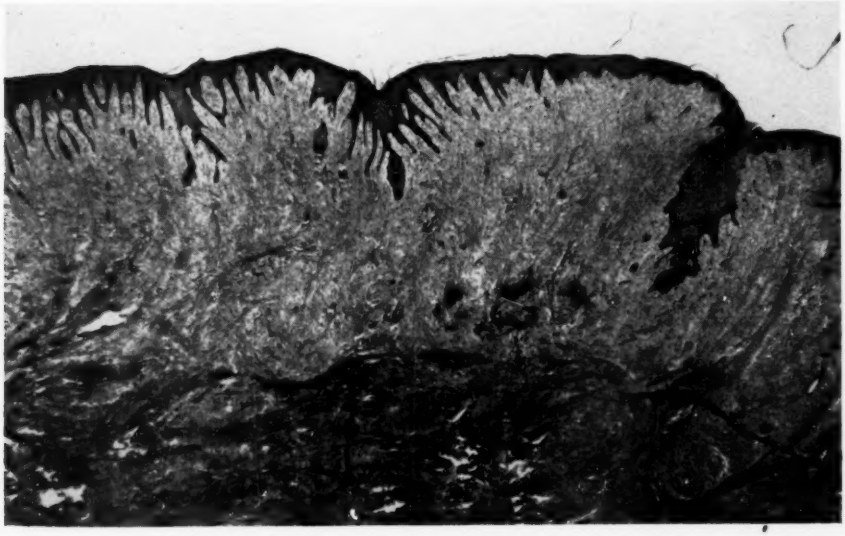

Fig. 6. Attached gingiva showing a scalloped appearance of the surface contour. This is the basis for the clinical description "resembling coarse orange peel" often given in these cases. Also note the deep epithelial folds and the epithelial pearl. The thickness of the epithelium appears reduced where it covers the height of the papillary ridges. The epithelial pegs are long, thin, and branched. H. & E. Magnification 40×.

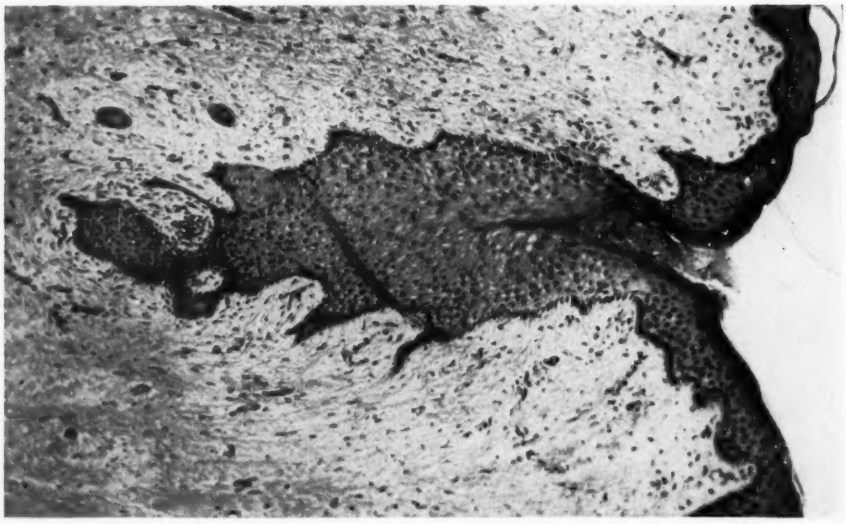

Fig. 7A. High power view of one of the deep epithelial folds, observe the small number of inflammatory cells. H. & E. Magnification 110×.

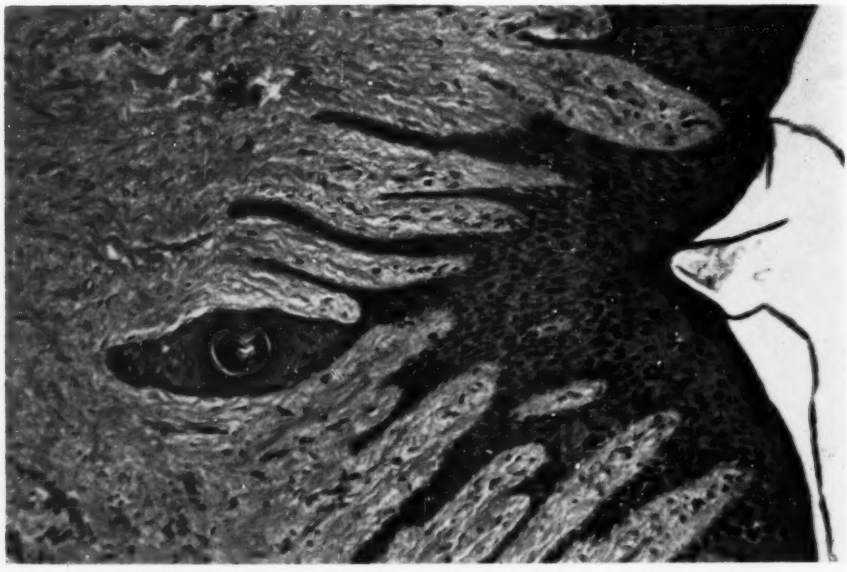

Fig. 7B. Another epithelial fold showing an epithelial pearl contained in an epithelial peg and few inflammatory cells. H. & E. Magnification 165×.

ridges appear thin, long and branched (Figure 6). In the epithelium of the attached gingiva are seen deep folds (Figure 7A). Some of the epithelial ridges contain epithelial pearls (Figure 7B). The papillary layer of the propria shows slight edema and infiltration with chronic inflammatory cells in the crevicular area. In the reticular layer, which is very thick in diameter, are seen numerous heavy collagenous fiber bundles; many of the fibers appear fragmented and possibly hyalinized in places (Figure 8). A small number of fibrocytes can also be observed in this layer; their nuclei are dense and shrunken. Some calcified bodies are also visible in this stratum (Figure 9). A mild degree of perivascular round cell infiltration is also present. None of the changes could be considered malignant.

The histologic findings in congenital gingival fibrous hyperplasia have been described in detail by many authors and were reviewed recently.[1,2] Inflammation is not an outstanding feature in these cases and may be entirely absent. The thin epithelial layer and the long, branching epithelial

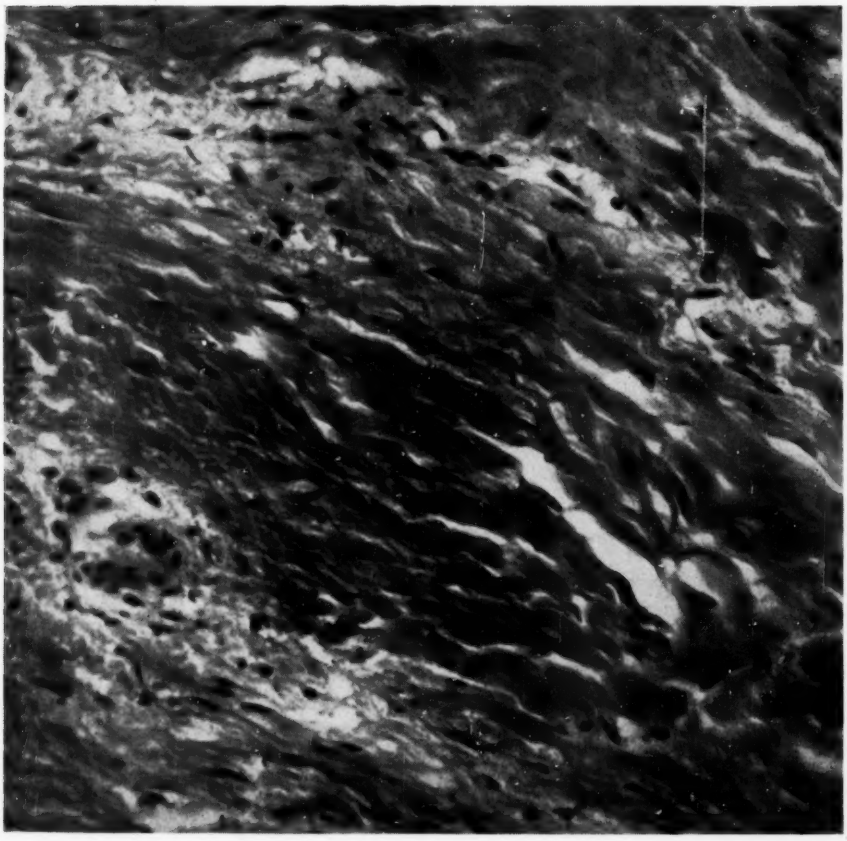

Fig. 8. Observe coarse fiber bundles in the reticular layer. Some of them appear broken up and hyalinized in places. Notice the small number of cells in the tissue, mainly fibrocytes with thin, dark, elongated nuclei. H. & E. Magnification 500×.

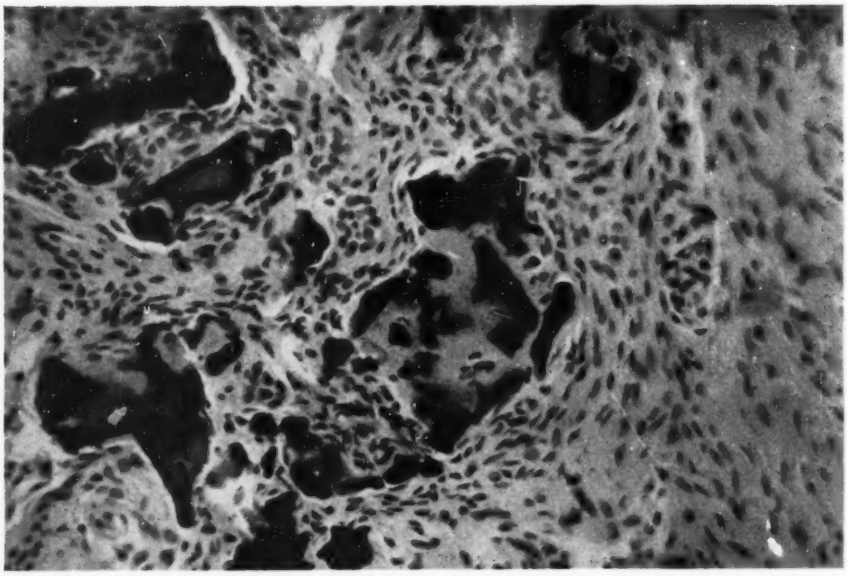

Fig. 9. Calcified structures in the reticular layer, possibly calcified thrombi. H. & E. Magnification 300×.

pegs are also considered characteristic, as is the extreme thickness of the reticular bundles. Indeed it is the hyperplasia of the reticular layer that is responsible for the extreme increase in total thickness of the gingiva, and probably also for some of the changes in the configuration of the epithelium. Among the features observed by us and less well documented in literature are deep epithelial folds;[9] these may be the basis for the exaggerated gingival stippling in these cases resembling orange peel. The epithelial pearls or whorls have also been mentioned.[10] The presence of calcified structures in the reticular layer, noted by us in many of these sections, has not been stated in literature as a frequent finding in this condition; however, a considerable number of routine gingival biopsies has been shown to contain such inclusions.[10, 11]

TREATMENT

The treatment consisted of: a) surgical removal of all excessive gingival tissue, b) extraction of retained primary teeth, and c) exposure of permanent teeth that were retarded in eruption.

The oldest child was scheduled for surgery after preliminary cleaning of the teeth. One-half hour before operation, she was premedicated with 1½ gr. seconal. Under regional anesthesia routine gingivectomy and gingivoplasty with the Radiosurg scalpel were performed in one quadrant of the mouth. Primary teeth were extracted and the overlying bone removed from the occlusal surfaces of the permanent teeth. The unerupted permanent 1st molar was uncovered and completely exposed. A surgical cement pack (slow setting) was incorporated into fine mesh gauze. Sutures were then placed through the buccal and lingual tissues and left untied. Then this surgical cement pack was placed over the operated area and the sutures drawn tight and tied. Operating one quadrant at a time, this same procedure was followed at one week intervals taking 4 weeks to operate

the entire mouth. Figure 4A shows the case before treatment and Figure 4B demonstrates the improvement six months after surgery.

This course of treatment was followed for two children. It is planned to follow this general procedure with the other children.

SUMMARY

1. A report of 6 siblings and their father afflicted with hereditary gingival fibrosis is presented.

2. The mode of inheritance in this family is apparently dominant. No other inherited abnormality was found to be present.

3. Histopathologic findings of the gingival tissues is presented. Presence of calcified structures in the reticular layer is reported and discussed.

4. Slight difficulties in speech, mastication and personality were caused by this condition. General health was found to be unimpaired and skeletal maturation was within normal range.

5. Eruption schedules of primary and permanent teeth were delayed, calcification of the teeth was found to be within normal range.

6. A method of treatment is briefly mentioned.

ACKNOWLEDGEMENTS

Authors wish to acknowledge the aid received from Miss Clare Holly for preparation of the biopsy specimens, Dr. Ellis B. Jump, Head of the Department of Anatomy, for the production of microphotographs and Mr. Russell F. Culbertson, Department of Audio-visual Aids for the other illustrations.

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Tooth Mobility*

The Measuring Method. Initial and Secondary Tooth Mobility

by HANS R. MÜHLEMANN, M.D., D.D.S.,** *Minneapolis, Minn.*

THE special morphology of the periodontal structures anchoring the roots of teeth in the alveolar bone allows for a certain physiological tooth mobility. *** From clinical experience we know that TM can vary. It is somewhat more pronounced on erupting teeth than in the healthy adult dentition. It increases in the presence of apical abscesses and during orthodontic treatment. Clasped teeth often are more mobile than other teeth in the same dentition. Increased TM may be one of the cardinal symptoms of periodontal disease. Several attempts, which will be described in some detail in the following review of the literature, have been made to evaluate quantitatively the degree of TM or tooth loosening. The most primitive recommendation, yet the only one to be found in the current textbooks of periodontia, is to test the amount of TM by moving the crowns between two fingers. For example, Miller (1950)[1] recognizes three levels of increased TM. Mobility No. 1 is the first distinguishable sign of movement greater than normal. Mobility No. 2 is a movement which allows the crown to move 1 mm. Mobility No. 3 allows the tooth to move more than 1 mm. Obviously, such classifications are insufficient, nonscientific, and highly subject to individual interpretation. If a more scientific method for quantitative evaluation of the degree of TM were available, many questions regarding TM could be answered. At present there is no information about the relationship between crown excursions and forces producing them or about the mechanism of TM. More specifically the following questions are unanswered: What are the differences of mobility between single and multirooted teeth, at different ages and under different functional conditions? How does TM rise on application of long lasting artificial local stress, such as during orthodontic treatment? Is there an optimal increase of TM for moving teeth orthodontically? What is the relationship of TM to occlusal trauma? How great an increase of TM is necessary before physiological TM becomes pathological? How does TM increase in gingivitis, periodontitis, periodontosis, and alveolar atrophy? What is the effect of pocket formation on TM? Is an increase of TM due to quantitative or qualitative modifications of the periodontal tissues? Are TM changes produced by systemic influences? Can alterations in TM be used to evaluate the benefit of local and systemic dietary and drug therapy? How do bridgework and splinting procedures affect TM? What changes in mobility are induced by removable partial dentures? A definite answer to these and similar questions would be of great diagnostic, therapeutic, and prognostic value in many fields of dentistry. It is the purpose of this and future investigations to provide an answer to these problems.

*This is the first of a series of reports about tooth mobility.

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***The abbreviation TM will be used to designate tooth mobility.

REVIEW OF LITERATURE

Elbrecht (1939)[2] measured TM with a large dial indicator that was fixed on a tripod in front of the patient's mouth. The dial indicator registered the labio-lingual crown excursions which were produced by digital pressure. The author points out the inaccuracy of the method, since movements of the head could interfere with the movements of the crowns. He gave a classification of increased TM. Only values over

0.75 mm. in total labio-lingual crown excursions could be determined with this method.

Werner (1942)[3] used an instrument that he improperly called an "oscillometer." It consisted of a rod with a scale attached and held on the front teeth. By moving the neighboring or proximal tooth labio-lingually with a force of 700 grams, the resultant difference of tooth position could be read on the scale. Differences of tooth position smaller than 0.25 mm. could not be evaluated.

Beyeler and Dreyfus (1947, 1949)[4,5] approached the problem of TM differently. Their vibrometer does not measure labio-lingual excursions of crowns but tries to determine the behavior of teeth when exposed to oscillatory vibrations of high frequency. These authors gave only preliminary laboratory results. No clinical reports were published.

The vibration technic was also used by *Manly et al.* (1951)[6], however with still higher and variable frequencies. Their reported preliminary clinical results were inconsistent.

Zwirner (1949, 1951, 1952)[7-9] studied axial TM on separated rat jaws with an electronic device. His method is highly sensitive. The probable cause for the lack of reports on human TM is the technical difficulties which have been encountered.

Cross (1951)[10] recently demonstrated a TM measuring device about which no details have been published to our knowledge.

Jung's (1952)[11] experiments were concerned with measurements more of the elastic deformation of the mandible during mastication than of TM of individual teeth.

Mühlemann (1951)[12-16] used intraorally attached dial indicators to determine the degree of crown excursions that were produced by known static forces. He reported extensively on the physiological and pathological TM.[14] His method was called periodontometry.[16]

Fig. 1. Forcemeter. When pushing or pulling with the leverarm (A) the forces used are registered in grams on the dial.

METHOD

With a forcemeter (Fig. 1) known static forces of different magnitudes are applied to the crowns of teeth at a right angle to their long axis. Forces ranging from 50-500 grams are directed labially or lingually for about two seconds. The instantaneous labio-lingual and linguo-labial crown excursions are measured with an intraorally attached dial indicator (Fig. 2). Its contact point (A) is placed perpendicular to the lingual or labial surface of the crown two to four millimeters from the incisal edge or occlusal surface. The contact point of the dial indicator when moved in or out by the crown during crown excursions sets the hand (B) on the dial in motion. The movement of the hand over the markings (C) on the dial indi-

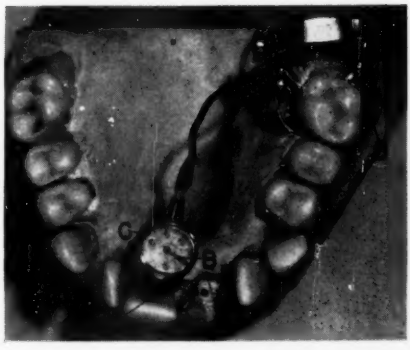

Fig. 2. Micro-dial indicator. (A) contact point, (B) hand, (C) dial markings, (D) rubber-dam clamp.

cator registers the excursions of the crown in hundredths of millimeters. The dial indicator is fixed by means of a rubber dam clamp (D) to a molar tooth. It is adjustable so that the contact point can be placed on that part of the tooth where TM is ideally measured. The question arises whether the tooth to which the holding apparatus is attached would also move and therefore invalidate the TM measurements of the tooth in contact with the dial indicator. The amount of force transferred from the tooth to be measured to the tooth or teeth anchoring the dial indicator depends upon the force necessary to depress the contact point into the micro-dial indicator. This force is less than five grams. Therefore, a transmission of forces greater than 5 grams to the anchoring teeth is impossible. After positioning the contact point slightly depressed on the lingual surface of the tooth whose mobility is to be measured, the position of the hand on the dial is noted. An example will be given: The hand may be resting on No. 32. The crown of the tooth is then pushed from its rest position "R" into a labial position "L_1" with a force of 500 grams. The position of the hand will move on the dial indicator from 32 to 14. This "L_1" position is recorded as 14. As soon as the force is released the crown and the hand on the dial indicator will return almost to the rest position (31="R_L"). From this position the crown is moved with the same force (500 grams) palatally. The hand position "P_1" may be 46. Upon the release of the force the hand moves again with the tooth, returning towards rest position, e.g. 33 ("R_P"). All the steps in the measuring procedure are made at equal time intervals which should not exceed three seconds. In order to substantiate the measurements, the labial and lingual excursions are repeated ("L_2", "P_2"). A typical record of these measurements would look as follows:

		R	L_1	R_L	P_1	R_P	L_2	P_2
1	500 Gm.	32	14	31	46	33	12	45

From such a record the following information can be calculated:

1. The labial excursion (L_{500}): 32 to 14 = 18
2. The palatinal excursion (P_{500}): 32 to 46 = 14
3. $L_{500} + P_{500}$ = 32
4. The uninterrupted total crown excursion (T_{500}) = 33

These numbers express TM in hundredths of millimeters when 500 grams are applied to the crown. If one is interested only in the total excursion, L + P and T are advantageously averaged: $\frac{32 + 33}{2} = 32.5$

MATERIAL

Two thousand TM measurements were made, with the method described, on seven Rhesus monkeys of different ages under intravenous Nembutal anesthesia. The dial indicator was attached by means of rods and self-curing acrylics to the cuspids (Fig. 3), or to the posterior teeth. The TM of erupting upper incisors, fully erupted upper incisors, upper bicuspids and molars was measured using forces from 50 to 500 grams. The data from these measurements were plotted graphically, the forces in grams being on the abscissa, the total crown excursions (T_{100}, T_{200} . . . . . T_{500}500) in hundredths of millimeters on the ordinate. When the plotted points are united, a *tooth*

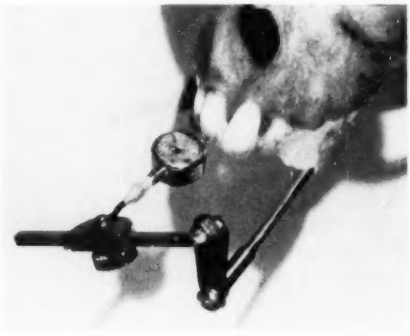

Fig. 3. Mico-dial indicator attached to monkey cuspid by means of rods and self-curing acrylics.

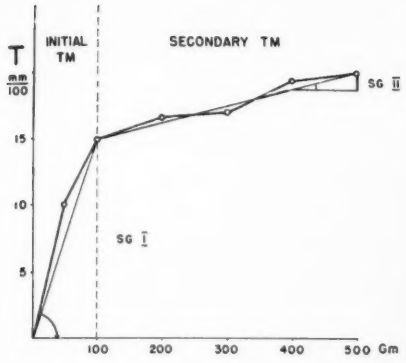

Fig. 4. Tooth mobility curve showing initial and secondary TM. (SG_I) Slope Gradient of initial TM. (SG_{II} Slope Gradient of secondary TM.

mobility curve results (Fig. 4). The monkeys which were used for TM measurements were sacrificed and serial sections were made from all the periodontal structures of the teeth on which TM had been determined. The specimens were cut labiolingually through the long axes of the roots. The sections showing the narrowest periodontal membranes on the labial and lingual side were photographed, and prints, enlarged 10-20 times, were made. On these prints the tips of the marginal bony crests were united with a straight line (Fig. 5, "A"). A second line ("B") was drawn parallel to this line and one millimeter apically to it. In other words, if the photograph was enlarged 10 times, the line on the print would be 10 mm. below the first line. The regions where the second line ("B-Level") crossed the periodontal membrane were identified under the microscope in the original histological sections and widths of the labial and lingual periodontal spaces and lengths of the roots were measured and compared with the TM measurements.

Fig. 5. "B-level" for determination of periodontal membrane width. In the original section the lines "A" and "B" are separated by 1 mm. (Monkey MF 1↓, magnification 7.5×).

RESULTS

TM curves obtained from teeth in different stages of eruption are assembled in Figure 6. Under normal conditions (Figs. 4 & 6) TM curves regularly show a rather steep initial and a subsequent shallow secondary slope. The initial slope, which is the graphical illustration of *initial tooth mobility*, passes into the secondary slope (*secondary tooth mobility*) when the forces producing the crown excursions are higher than 50 to 100 grams.

In order to define steepness and shallowness of the slopes in these curves, it is necessary to use *slope gradients* (SG). A slope gradient indicates how many hundredths of millimeters the crown excursions in-

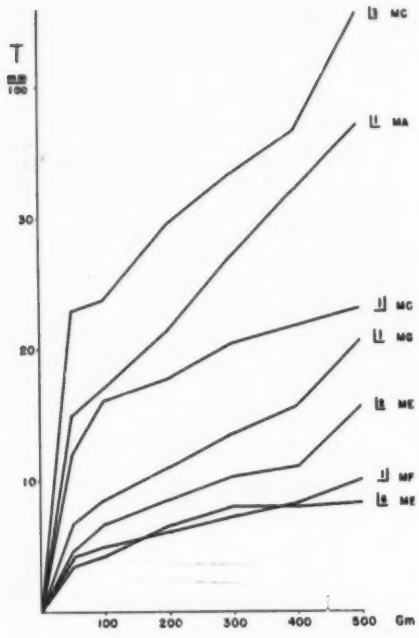

Fig. 6. TM curves of different teeth from different monkeys. MC $\frac{3}{}$ a cuspid in its initial stage of eruption, MA $\frac{1}{}$ an incisor further erupted than MC $\frac{3}{}$, MC $\frac{1}{}$ an incisor in the terminal stage of eruption, MG $\frac{1}{}$ posteruptive stage, but still in mixed dentition, ME $\frac{2}{}$, MF $\frac{1}{}$, ME $\frac{6}{}$ teeth in posteruptive stage and under heavy function.

crease within a range of 100 grams of applied force; e.g. the curve of the tooth in Figure 4 has a slope gradient of 15 for its steep portion of initial TM (TM_{100}), against a slope gradient of 1.25 for its shallow portion of the secondary TM $\frac{(T_{500} \text{ to } T_{100})}{4}$. In Table I are listed average slope gradients resulting from 100 TM curves of erupting anterior teeth of young monkeys and of well functioning incisors and molars of old monkeys. From this Table it can be seen that:

1. both single and multirooted teeth present the same fundamental pattern of TM, illustrated by an initial and a secondary phase in the TM curves.

2. the slope gradient of the initial TM is always higher than the slope gradient of secondary TM.

3. initial and secondary TM decrease with the functional aging of the teeth.

4. the slope gradients of molars are slightly smaller than those of the incisors.

Comparing the TM measurements with with the histological sections, the following observations were made (Table II): *Initial* TM is relatively high in the presence of wide periodontal membranes (MC $\frac{3}{}$, MA $\frac{1}{}$). However, initial tooth mobility is not just determined by the width of the periodontal membrane alone. Comparing all the data from the other monkey teeth in Table II one can find that the width of the periodontal membranes is *increasing* and at the same time the initial TM is *decreasing*. This fact is due to differences in the histomorphologic structure of the periodontal membrane. In Figure 7 the histological sections from the same periodontal region of upper right incisors, MC $\frac{1}{}$ and MF $\frac{1}{}$, can be compared. The widths of the periodontal spaces are different and there is a dissimilarity in the arrangement and density of the periodontal membrane fiber bundles. The density is more pronounced in the specimen from MF $\frac{1}{}$ which had a wider periodontal membrane and a shorter root but a smaller initial TM than MC $\frac{1}{}$. Monkey MF had a well functioning adult dentition, whereas monkey MC was only in the stage of mixed dentition.

DISCUSSION

This study has revealed information about the relationship between instantaneous crown excursions and static forces applied horizontally to the crowns. In order to initiate labial or lingual displacement of a tooth from its physiological rest position only small forces (50-500 grams) are required. The resistance of the tooth supporting structures against a displacement of the root is low at the initial phase of the crown excursion. It is definitely

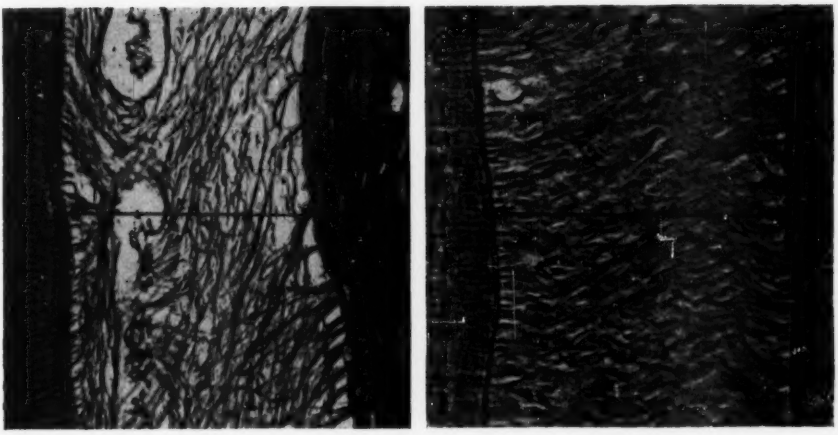

Fig. 7. Periodontal membranes of different widths and structure from the same area of upper right incisors (magnification 330×). Specimen MC $\underline{1}|$. Membrane width 0.16 mm. Intial TM (T_{∞}) 0.12 mm. Specimen MF $\underline{1}|$. Membrane width 0.20 mm. Initial TM (T_{∞}) 0.035 mm.

higher in a subsequent secondary phase when one tries to produce (with larger forces) a still greater crown excursion or a further displacement of the root. In view of this fact, a grouping into initial TM and secondary TM becomes evident. Both are represented in TM curves by different slope gradients.

The present results correspond to the findings in a previous report on 4000 measurements of human TM,[14] where it was found that the slope gradients of the secondary TM do not decrease significantly until the applied labio-lingual forces reach 1500 to 1750 grams. However, within the range of these latter forces a point is attained where the crown excursions cannot be increased any more without producing serious pain. This portion of the TM curves was called the region of the *terminal* or *maximal tooth mobility* (T_{max}) and it is so characteristic that reliable TM measurements could be performed without the use of a forcemeter. The crowns simply were pushed and pulled with a suitable instrument (scaler) labially or lingually until the region of terminal TM was reached. Measurements performed in this way, twice, and at time intervals of 30 to 50 minutes, on the same human incisors showed surprisingly small average differences between first and second measurements ($\triangle \overline{x} = + 0.25 \pm 0.11 \frac{mm}{100}$) demonstrating the reliability of T_{max} measurements. Using

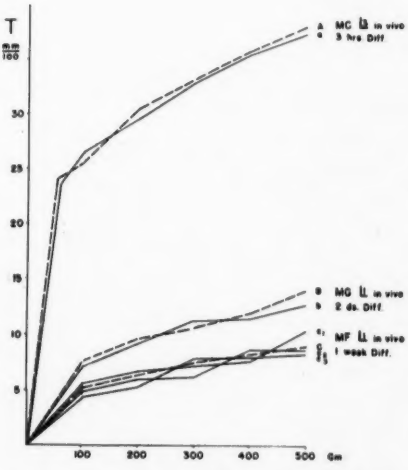

Fig. 8. TM curves of different teeth obtained at different time intervals. Time interval between "A" and "a"—3 hours. "B" and "b"—2 days. "C" and "c_{1-3}"—1 week.

TABLE I

Average Slope Gradients of 100 Tooth Mobility Curves

		Initial TM Slope Gradient	*Secondary TM Slope Gradient*
Anterior Teeth	Initial stage of eruption	34	4.3
	Terminal stage of eruption	16	1.8
	Post-eruptive stage	7	1.4
Molars	Terminal stage of eruption	12.5	1.9
	Post-eruptive stage	5.2	0.9

known forces as in the present report, the reliability of the dial indicator method is still better.[14] At time intervals of 3 hours, 2 days, and 1 week, the mobility of the same teeth of three different monkeys was measured. The TM curves constructed from these data are almost superimposable (Fig. 8).

The importance of a correct and standardized measuring procedure for the assessment of TM changes has to be emphasized. The consequence of different and improper adaptations of the dial indicator to the crowns has been reported in detail.[14]

The magnitude of TM has often been related to the thickness of the periodontal membrane.[6] Such a conception considers the amount of TM merely as a mechanical problem. Kellner[17] and Kronfeld[18] described the influence of function on the width and the morphology of the human periodontal membrane. In good function this membrane is wide and dense, it is small and loosely structured when less functioning. Our membrane width measurements are in accordance with their findings (Table II). Comparing these measurements with the corresponding TM values, it appeared that the histomorphological structure of the periodontal membrane is determining the degree of initial TM and that the width of the periodontal membrane is of secondary importance. One can find high

TABLE II

Initial Tooth Mobility, Periodontal Membrane Width and Intra-alveolar Length of the Root

Designation of Monkey and Tooth	*Stage of Eruption*	*Average Initial TM T_{50} mm/100*	*Width of Labial Plus Lingual Periodontal Membrane* mm/100*	*Length of Root mm/100*
MC|3	initial	23	81	8.3
MA|1	intermediary	17	74	5.7
MC 1|	terminal	12	49	10.2
MC|1	terminal	11	56	10.0
ME 1|		7.5	55	8.8
ME 2|		9.5	63	9.5
ME|1	teeth in	6.5	60	8.3
MF|1	heavy	5	60	8.7
MF 1|	function	3.5	61	8.2
ME|6		4.75	61	6.2
ME|2		4.75	77	9.9

*At B-level.

initial TM values but small periodontal widths. In such cases the periodontal membrane is poorly structured. On the other hand small initial crown excursions are associated with relatively large and densely structured membranes (Table II). These findings indicate the importance of the biofunctional qualities of periodontal structures on the degree of TM.

SUMMARY AND CONCLUSIONS

I. Tooth-Mobility measurements performed on monkey teeth by the use of an intraorally fixed micro-dial indicator and by the application of short lasting static forces to the crowns showed the existence of an initial and secondary phase of TM. This phenomenon is due to differences in the resistance opposed by the periodontal structures to an instantaneous labio-lingual moving of crowns.

II. Initial and secondary TM were found in single- as well as in multirooted teeth.

III. Erupting teeth had a greater initial mobility than erupted teeth. Initial TM was lower in well functioning teeth.

IV. The histomorphological structures rather than the width of the periodontal membrane influenced the degree of initial TM.

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An Evaluation of Factors Determining Prognosis in Inflammatory and Retrogressive Periodontal Disease: A Series*

by RICHARD WILLIAM SLATTEN,** D.D.S., *Richmond, Va.*

THE periodontides include all diseases of an inflammatory nature affecting collectively the various components of the periodontium. A periodontitis, in contrast to a gingivitis, is characterized by its involvement not only of the gingiva but also of the alveolar bone and corresponding parts of the periodontal membrane.

Periodontitis simplex and *periodontitis complex* constitute two distinct groups of periodontal pathosis characterized by inflammation. Insight into the basic nature of these two periodontal involvements will lead the student into a rational concept of their prognoses.

An evaluation of any case of *periodontitis simplex* must give due consideration to the pathogenesis of gingivitis, for periodontides of a simplex nature are always secondary to any one or more of the aforementioned types of gingival inflammation. Periodontitis simplex is, then, nothing more than a (late) development in the constantly progressive phenomenon of gingival disease. The point in time wherein gingival disease becomes simplex periodontal disease may be a matter of much debate among clinicians. However it is an irrelevant point for such oral pathosis must be regarded as a broadly moving and progressing panorama. All disease should be considered as the summation of extremely complex processes seldom, if ever, portraying clear-cut demarcations in the phases of its development.

An evaluation of any case of *periodontitis complex* must take note of the retrogressive disorders of the periodontium, to one or more types of which periodontitis complex is always secondary. Thus the basic and initiating pathosis of periodontitis complex is retrogressive in nature. It is vital that this line of reasoning, this distinction, be followed in the evaluation of inflammatory disease of the periodontium. Forbus says: "In studying human disease it is rare that a single or simple injury is to be dealt with. Because of this, it is necessary to distinguish between a primary injury and those injuries which may develop subsequently."[10] The application of this dictum is exemplified when we denote complex periodontitis as a retrogressive disease clinically highlighted by a subsequent, superimposed picture of inflammation.

The importance of recognizing the course followed by any disease process has already been emphasized. Some aspects of the course of inflammatory periodontal disease can be identified at this time.

The ultimate result of all inflammatory periodontal disease is the breakdown of the tooth's supporting structures until such time that it must be removed or of its own accord exfoliates. Both the rate at which this occurs and the course by which it is accomplished vary widely. The innate nature of the processes at work produce a variety of symptoms now well known, although the exact mechanisms by which these ends are accomplished, along with all the factors which aid and abet them, are not now fully understood.

Simplex periodontal disease might be thought of as a moving process of inflammatory disease which, directionally, follows a crown-to-root course. In other words, beginning with gingival disease progressively deeper involvements of the support-

*The initial installment of this series published in the Jour. Periodont. 24:252 (1953) was devoted to a discussion of prognosis in gingivitis.

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ing structures of the tooth ensue through extension of the inflammatory processes at work. As inflammation alters the integrity of structure, so in turn does alteration of function result. The destructive cycle created will perpetuate itself unto the disastrous results so often evident clinically if it is not interrupted.

On the other hand, complex periodontal disease might be thought of as pursuing a root-to-crown course, i. e., its pathosis, basically retrogressive in nature, begins in the deeper periodontal structures and at some undetermined time a clinical picture of gingival inflammation becomes superimposed. Just when and how this final clinical picture develops forms the basis for much speculation. As severe retrogressive changes occur in the bony supportive structure of the tooth, mobility becomes marked. The tension or strain which this increased mobility may place on the epithelial attachment contributes, perhaps, to a pathological deepening of the sulcus. That deepened sulci sooner or later become sites of increased bacterial activity is a common observation.

The prognosis of any given case of periodontal disease stems from the infinite number of features which characterize it and a definitive prognosis can only result from careful analysis of all its salient features. So much excellent material fills the literature devoted to special problems offered by individual types of periodontal cases that the scope of this paper has been narrowed to provide for only an attempt at generalizations.

In the light of what has been said about simplex and complex periodontitis, some organized thinking regarding prognosis should materialize. The basic nature of each process is known and the most striking feature of the course of each has been drawn. Several points of interest should now be clear and they may be summarized briefly as follows:

(1) The initial and basic pathosis in simplex periodontal disease is inflammation, a disease process about which knowledge is rather extensive;

(2) the initial and basic pathosis in complex periodontal disease is retrogression, a disease process the background knowledge of which is comparatively sketchy;

(3) the course of simplex periodontal disease begins in the gingivae where inflammation gives rise to a variety of subjective and objective symptoms; and

(4) complex periodontal disease runs a course initiated in the deeper supportive structures of the tooth and only after considerable damage to these structures has occurred will symptoms likely come to light.

On the basis of these observations we may say that, all other factors being equal, simplex periodontal disease usually offers a better prognosis than does complex periodontal disease. In the last analysis, any disease process which is characterized by ostensible signs and symptoms early in its course is more apt to be detected early and, consequently, treated (early) while a minimum number of irreversible tissue reactions are in progress.

B. RETROGRESSIVE PERIODONTAL DISEASE

Retrogressive change, a degeneration, involution, or atrophy of tissue, has concerned pathologists for a great many years and has recently received considerable attention from gerontologists. The idea has evolved that inflammatory change is really physiological in nature (being, as previously stated, ". . . functional tissue responses for protective and regenerative purposes"[8]) and it is within the realm of possibility that retrogressive change may come to be viewed in a similar light. If so, it may follow that inflammation, generally, is perhaps a characteristic response more normal to young tissues while retrogression is perhaps more characteristic of ageing tissues. If disease is "the abnormal response of living cells and individuals to environmental stimulation,"[11] perhaps we should view with more alarm and foresee a less favorable outcome for those cases of disease wherein retrogressive change is evident in the young and inflammation is superimposed on senile tissues undergoing retrogressive change.

Retrogressive change in the periodontium is designated, in the classification here followed,[12] by the term *periodontosis*. The periodontoses have been subdivided into three groups: *atrophic periodontosis*, *hyperfunctional periodontosis*, and *periodontosis gravis*. The tissue changes resultant in all periodontoses are those characteristic of morbid tissue degeneration rather than inflammation. The identification of the etiology pertinent in each case is usually a difficult one, and may, not infrequently, be impossible. This one fact alone poses such a problem that the prognosis of any periodontosis is rendered questionable. The chance of successfully removing an etiological agent which one cannot identify is probably negligible.

1. ATROPHIC PERIODONTOSIS

The atrophic periodontoses may be classified as *presenile* or *senile*. Such an approach to atrophy of the periodontium necessitates some brief observations regarding the changes normal to ageing tissues.

"The processes of ageing are to be distinguished from age changes. They are the underlying alterations of which the age changes are the manifestations. A great many age changes may be described but the processes of ageing are in the background almost wholly hidden from us."[7] These age changes in the masticatory apparatus are all intimately related to changes in structure and function and are, moreover, interrelated as structure-functional changes between the teeth and the periodontium and between the various components of the periodontium itself.

A detailed discussion of age-changes in the teeth themselves is beyond the scope of this paper. Suffice it to say that the hard dental tissues and the pulp are structurally involved as the organism ages and at least one of these alterations (occlusal wear) is of prime importance to the periodontium.

Age-changes in the periodontal membrane might be expected to parallel changes due to ageing in other connective tissues. Dummett states that "in the periodontal membrane, modifications associated with ageing include a gradual retardation of mitosis, a lessened capacity for cellular accretion, cellular atrophy, degeneration, increased cell pigmentation and fatty infiltration."[9]

Cementum seems normally to increase with age,[3] and the interdental alveolar crests seem normally to resorb with age.[4]

Ageing in the epithelium appears to be characterized by apical proliferation of the gingival attachment and shortening and rounding of the interdental (gingival) crests.[5]

These changes, as previously stated, are imperfectly understood and it is very difficult at this time to separate them in our thinking from changes in structure and function initiated by processes now convenient to label pathological. Alterations of a pathological nature seem frequently to accompany, perhaps modify in degree, and probably to some extent control changes intimate with ageing. "It would be nice to be able to distinguish natural death of cells following uncomplicated ageing from death due to other factors such as injury and disease."[6] Such a distinction is now extremely difficult to make. When this can be accomplished readily, insight will be gained into that vast range where the normal and the pathologic (seem to) overlap and their values merge.

The maintenance of normal structure where normal function is subminimal is unlikely if not impossible; that is to say, hypofunction is no more to be desired than hyperfunction. The periodontal membrane changes readily under varying conditions of function and, as regards the alveolar bone, "the trabeculae are formed in accordance with the requirements placed on them by normal or abnormal masticatory stress."[4] If such is the case, health is not likely to remain a characteristic feature of periodontal tissues which experience continuous dysfunction, e. g., in a so-called "open-bite" anterior relationship. Such ob-

servations are the basis for a classification designated *presenile periodontal atrophy*.

Senile atrophy is, on the basis of changes which seem to occur as ageing develops, a physiological change. There is, however, great variance in the rate at which such atrophy occurs and considerable difference in the degree to which it should be evident at a given age. Senile atrophy obviously constitutes one of those innumerable "ranges" which constantly confront students of health and disease.

The evidence of *presenile atrophy* always offers a poor prognosis. In very young individuals it is likely to be an expression of underlying general debility (metabolism) of tissues or of chronic disease.

The prognosis of *senile atrophy* is good or bad in proportion, seemingly, to the rate at which it occurs. The more rapid its apparent rate the less favorable is the prognosis for maintenance of the teeth.

2. HYPERFUNCTONAL PERIODONTOSIS

Any discussion of degenerative periodontal disease due to hyperfunctional stresses keynotes a consideration of the term "traumatic occlusion." This appellative was introduced in 1917[16] and the many features of the concept it denotes have been argued vehemently since that time. The relative merit of the term, traumatic occlusion, has previously been evaluated and the tendency is increasing to employ nomenclature more accurately descriptive of the fundamentals involved, e. g., traumatogenic occlusion or pathogenic occlusion.

So many schools of thought on occlusal traumatism have evolved over the years that it would be an endless task to discuss the merits of each and the relation to prognosis which each has been allocated. To formulate any rational concept of traumatogenic occlusion necessitates an evaluation of the "normal" occlusion. From time to time the many fields of dentistry have defined the innocuous occlusion as it concerns them directly and that of the periodontist is usually taken to be: that relationship wherein the stresses applied to teeth in function are within the bounds of tissue tolerance.

The appropriate role which should be assigned traumatogenic occlusion in the over-all picture of periodontal disease is still fluxional. Divergent opinions among well-known periodontists attest to the state of confusion which exists generally. While one writer[15] looks upon traumatic occlusion as a symptom of periodontal disease and not the (sic) principle etiological factor, another[14] "feels that traumatic occlusion can be a basic cause of periodontal disease." To make confusion rife we note in addition that traumatogenic occlusion has been pictured as "a local predisposing factor in periodontal disease,"[2] while still another author has described occlusal trauma as the primary cause of periodontal inflammation (sic) in healthy mouths only if the stress is sudden and excessive.[1] Through what channel might one direct effort in order to realize some degree of satisfaction from current opinion?

The effects of functional stimulation in the development, maturation, and maintenance of the structure of the periodontium is so fundamental as to command attention. No consummate conception of periodontal health or disease is possible in the absence of these essential details. While the observation that bone which is not functioning tends to become less dense is a common one and suggests that the structure of bone is in some manner actively related to its function, the exact explanation behind this phenomenon seems not as yet to have been fathomed. Moreover, bone responds to increased function. Under such circumstances changes may result in the form and structure of bone. That these changes must now be looked upon as symptoms of some fundamentally basic changes whose exact nature research is still seeking distinctly limits the scope of clinical activities, and that as yet full knowledge is not a realization

constitutes in no small measure the essence of present-day confusion.

Some perplexity might well be expected, however, for "it is not always easy to determine what is physiological or normal and what is pathological or abnormal in the matter of stimulation. What is harmful under certain conditions may be quite harmless or even beneficial under other conditions."[10] On no subject more than on this matter of traumatogenic occlusion is such reasoning applicable and it should serve to direct our interest away from occlusions as Angle classified them, with no intent to minimize the importance of his work but to channel thinking towards the consideration of occlusion and its biomechanical rôle in alterations in structure and function.

Any occlusion is a traumatogenic or pathogenic one if it has produced or is (capable of) producing injury to the periodontal structures. Since disease is associated with interrelated changes in both structure and function (changes in structure ⇄ changes in function) it seems plausible to anticipate pathogenic occlusions as both a cause and an effect. In a clinical sense we see this wherein a pathogenic occlusion produces structural change in the periodontium (periodontosis) and conversely wherein inflammation of the periodontium, giving rise to extrusion, drifting of teeth, etc., results in an occlusion's becoming pathogenic by virtue of an overt change in tooth relationships (periodontitis simplex). There will continue to be a great deal of general confusion about the pathological occlusion until the time arrives when it ceases to be regarded as some strange entity apart and immune from licensed concepts of physiology and pathology. An occlusion is a dynamic thing and its ability to function harmoniously may change theoretically from day to day and depends not only on the changing structure of its own components but as well on the interplay of structure and function in the periodontium which supports it.

The process of evaluating, from a periodontal standpoint, any given occlusion has unfortunately not been standardized. It should be realized from the onset that an occlusion which is giving rise to injury or pathosis, one which is pathological, may be responsible for tissue reactions varying considerably in nature and degree. If the nature of the pathosis resulting is severe, to the point that a distinct pathological entity such as *trauma* results, then that occlusion is quite logically a *traumatogenic* one. If, on the other hand, an occlusion is not producing this maximum degree of injury (trauma) by virtue of its hyperfunctional stresses, it can quite readily give rise to tissue changes less dramatic than those of trauma, ones retrogressive in nature. When such is the case, the occlusion involved ought properly to be labeled *pathogenic* rather than traumatogenic. Lyons suggests that these changes might well be described as "retrogressive tissue metaplasias."[13] It might be added, in order to emphasize a point, that whereas the term, pathogenic occlusion, may properly indicate one responsible for injury or pathosis of varying nature and degree, the term traumatogenic is distinctly limited in its connotation.

There are a number of signs and symptoms which have been described as characteristic of occlusions whose forces are no longer within the supporting tissues' tolerance. While the treatment of symptoms is often the only recourse it should be remembered that therapy so solely founded is inclined not only to be enigmatic but fallible in its results, a situation demonstrating again the importance of unceasing efforts to determine the underlying, basic causes of disease. The more disease is dealt with the more strikingly is illustrated what a tremendous handicap is created by a lack of knowledge about these basic causes.

The time has not yet arrived in which extensive generalizations can be formulated concerning the periodontal aspects of occlusion but there are a few general principles now accepted as fact by a large proportion of students in the field. Paramount among these is the contention that dental suppor-

tive structures are better able to withstand forces which are exerted in a direction parallel to the long axis of the tooth than they are those which approach an angle of 90°. The implications of such thought lead to the periodontist's concern, regarding the teeth in relation to their supportive structures, with a clinical evaluation of reduction of cusp height or its failure to occur. As cusp heights are reduced, and consequently stresses at right angles to the tooth, proportionately less work (resistance) is expected of the periodontium. As age-changes alter the periodontium this simultaneous reduction in cusp height appears desirable in order to maintain a balance between function exercised and altered structure's ability to meet with it.

This would *seem* to indicate that young periodontiums need to be endowed with a robust nature of unknown degree if they are to withstand the forces exerted by young teeth the nature of whose stress is in a measure due to their steep incline planes. Further, senile supportive tissues, characterized by structural changes, some physiological and some undoubtedly pathological, may suffer severe breakdown if abrasion has not altered the type of function (force) they must withstand.

The prognosis of cases whose causative factors include pathogenic stresses is varied. For the most part one may anticipate successful removal of pathogenic factors where they are primarily an effect (though, to be sure, they proceed to act in a causative rôle), as in advancing gingival disease. Here, instigation of a new balance between structure and function will usually induce a satisfactory return to health. The outlook for cases in which a pathogenic occlusion is primarily a causative agent may not be so favorable. In such instances it is probable that lowered resistance of tissues is an important consideration. Such cases present tremendous problems. Bone and connective tissue physiology are extremely complicated and the incomplete understanding of disease processes in these tissues makes the results of therapy questionable. In the majority of cases one can only direct the patient into the hands of competent medical care, balance dynamic forces as well as possible, restore function where it has been lost, distribute the forces of mastication over as great a working area as possible, reduce stresses to a minimum on individual teeth, induce health in the supporting structures involved (to the extent possible), and then hope that an equilibrium between stress and resistance will be achieved. Until such time as new evidence denotes more clearly what therapeutic measures are indicated to reverse the basic tissue changes involved, the prognosis in such cases will continue to be not only indecisive but largely a matter of individual clinical judgment alone.

3. PERIODONTOSIS GRAVIS

Periodontal disease, produced primarily through general systemic disease, will here be discussed under the title of *periodontosis gravis*.[12] Students in the field of periodontology have for a very long time concerned themselves with the question of local etiology and systemic etiology as regards periodontal disease and the result has been an amassment of heterogeneous ideas in the literature. The use of language on this point has been rather disorganized in the past. One might give close attention to the schools of thought which have developed.

Since the definition of terms is a rather fundamental consideration it might be wise to give some space to a discussion of the terms "local in etiology" and "systemic in etiology." Just what do these terms and variations of them signify in relation to periodontal disease? "Local in etiology" would seem to mean simply that periodontal pathosis is or may be caused by external, locally acting factors which, by giving rise to irritation (injury), effect a resulting state of disease by their very presence in, on or near the periodontal structures. In attempting to define "systemic in etiology," two avenues of approach to the problem are open. It might be taken to mean that systemic disease (dysfunction?), of one

sort or another, is able in itself to be the agent which brings periodontal disease about, i. e., it may *cause* periodontal disease. From another point of view one might define "systemic in etiology" by recourse to another term: that of *tissue predisposition*. One thus implies that systemic disease, while not in itself able to cause periodontal disease, so predisposes the periodontal tissues (or alters their intrinsic ability to maintain a state of health) that they fall heir to a state of disease under the influence of *exciting factors* which might not ordinarily be expected to provoke in them disease of a given intensity. How are we to go about fitting such a concept of etiology into present-known patterns of disease?

Man may be somewhat singularly outstanding when considered in relation to his environment, both natural and self-created. He is a biological mechanism carrying on his life processes while continually exposed to an overwhelming number of agents (chemical, mechanical, physical, and psychic) potentially capable of inducing a state of disease in him. Thus health itself may be in part the act or state of action wherein disease (or the causative agents of disease) is resisted. It would seem that the various "defense" mechanisms of the individual must function unceasingly in order for health to be a realization. When one considers microorganisms alone it seems clear that bodily defenses must hold countless times in the face of attack. One might deduct this from evidence indicating that organisms now thought responsible for certain phases of oral necrotizing disease may often be recovered from mouths almost completely healthy (clinically). The question of whether or not disease, of bacterial nature for instance, results in a given situation is of course rarely dependent on any one single factor. While the number and virulence of the organisms is a vital consideration nevertheless the resistance of the host must be taken into account. With the possible exception of simple traumatic injuries there seems no other way at the present time to explain satisfactorily certain disease states than to assume as fact the concept that disease is a ways the result of a change in balance between the resistance of a tissue (or tissues) and the potential ability of extrinsic factors to excite pathosis in it. In other words, disease will follow when the resistance of tissues *is* below or *falls* below certain optimum levels since: (1) it can no longer exercise its regular function, and/or (2) it can not face the insult of extrinsic factors brought to bear on it.

Exactly what now is the gist of this long-argued problem of local etiology and systemic etiology in periodontal disease? It amounts to the fact that one school of thought has declared the etiology of all periodontal disease to be purely local in origin; another, the idea that all periodontal disease is totally systemic in causation. Thirdly, the idea has been advanced that periodontal disease may in some instances be local and in some instances systemic in its causation. A final elaboration of past concepts has culminated in the now widely accepted thesis that *all* periodontal disease is at one and the same time, though to greatly varying degrees, local and systemic in its etiology.

What then is the basis for a classification such as *periodontosis gravis* if all periodontal disease is at one and the same time both local and systemic in its causation? It will be seen that the need for it has grown up as the meaning of what constitutes "systemic in etiology" has been more fully defined. Where states of retrogressive disease become evident in the periodontium due, *primarily*, to general systemic disease, the case should be especially categorized since the direction its treatment takes and the tone its prognosis assumes stem more or less directly from the general nature of the systemic disturbance. In such cases the dentist will probably to no avail remove or eradicate all those factors acting locally to provoke pathosis which we now recognize and are pleased to denote as "irritants." But the point to be recognized in this instance is that biological tissues or structures must enjoy their intended function (or at

least some ill-defined degree of function approaching total capacity) if health is to prevail, and where this intrinsic resistance of which we have spoken is materially lowered through an over-all dysfunction generally, then, in these cases, even normal function may assume the nature of "irritation." For example, a given bone may function quite happily when the over-all metabolic processes of its host are conducive to its welfare and the integrity of that bone will remain unaltered when no external factors capable of provoking injury are encountered. However, if the balance of general metabolic activity is disrupted, as for example in osteomalacia (osteoporosis), then that bone may suffer adverse changes in the course of normal function. So-called "pathological fracture" is an example of this metamorphosis of function.

Logically, more and more cases of retrogressive periodontal disease might be expected to find their way into the category of *periodontosis gravis* as methods of detecting general systemic disease (dysfunction) are refined. Cases are often so consigned now when exhaustive treatment fails. Since erring is possible by even the most competent clinicians, an incorrect diagnosis might be considered a possibility in cases where little or no apparent response to therapy is forthcoming.

As regards prognosis in cases classified as *periodontosis gravis* few general conclusions can safely be drawn. If a medical survey of the total individual reveals findings of a positive nature, ones which point to disease or dysfunction which falls within the scope of knowledge about departures from health now understood, then the outlook for the periodontal tissues will be as favorable as for any other tissue which may suffer within the scope of that dysfunction. Findings of a negative nature usually lead to the frustration incident to most empirical treatment. Nevertheless, it is not unreasonable to suppose that lack of knowledge in this particular realm of endeavor will ultimately prove to be the rung utilized in another step upward toward the goal of full control by man of himself.

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Repositioning the Attached Gingiva*

by CLAUDE L. NABERS, D.D.S., M.S.D., *San Antonio, Texas*

WHEN the base of a periodontal pocket has shifted so far apically that it is beyond the muco-gingival junction,[1] it creates a difficult treatment problem. The difficulties which arise are due to the structural differences between attached gingiva and alveolar mucosa. The attached gingiva has a very dense collagenous, fibrous connective tissue, firmly attached to the underlying tooth surface and bone. There are no elastic fibers in this tissue. The alveolar mucosa, on the other hand, has a loose submucosa which is rich in elastic tissue. The elastic fibers will cause this tissue to retract, leaving a gaping wound after surgery. There is also a difference in the surface epithelium between the two areas. The attached gingiva is covered by a heavy stratified squamous epithelium with long epithelial ridges and is well keratinized on the surface. The epithelium of the alveolar mucosa is rather thin; has no ridges and is non-keratinized. This structural difference is due to functional differences.[2] The attached gingiva is adapted to the function of mastication, whereas the alveolar mucosa has the function of lining the oral cavity, keeping it moist and smooth. Its surface cannot well withstand the friction of food during mastication.

Three diagrams will illustrate the arrangement of gingiva, sulcus, muco-gingival junction under normal conditions (Fig. 1), in a case with a deep pocket but with its base still within the attached gingiva (Fig. 2), and a case where the base of the pocket passed the muco-gingival junction and extended into the alveolar mucosa (Fig. 3). Such periodontal pockets, of course, go hand-in-hand with resorption of the alveolar crest.

If, in a case like that illustrated in Figure 2, a gingivectomy has to be performed, the incision can still be made within the attached gingiva and one will encounter no special difficulties in healing or prognosis. However, if the bottom of the pocket has passed the muco-gingival junction (Fig. 3), the line of incision will be in the alveolar mucosa. As a result of the incision, the wound will gap widely and will be painful. The healing will be retarded because of the mobility of the tissues. Even more important, the newly-established gingival margin will be covered by a tissue

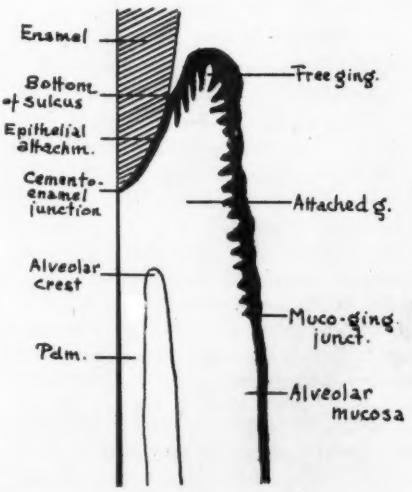

Fig. 1. Diagram, illustrating "normal" relation of gingiva, sulcus, epithelial attachment and alveolar crest.

not adapted to the functional requirements. Injuries in mastication will occur. Proper brushing may be difficult and painful with the result that oral hygiene cannot be well maintained. The prognosis will be at least doubtful.

In encountering such a case, an attempt was made to overcome these difficulties.

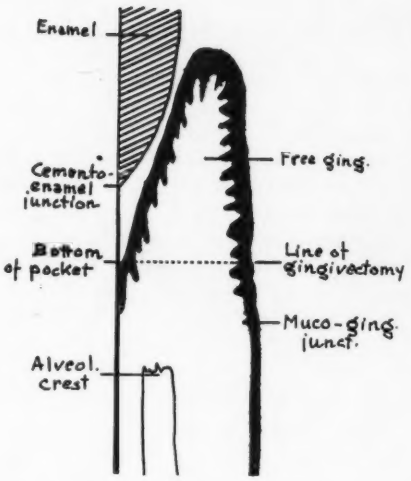

Fig. 2. Diagram, illustrating periodontal pocket—the base of which does not reach below the muco-gingival junction.

Instead of a gingivectomy, a flap was made by a vertical incision mesial to the area of the deepest pocket. The flap was retracted and the alveolar crest and pocket area exposed. All granulation tissue was eliminated; the inside of the flap cleaned, removing all granulation tissue; the gingiva trimmed

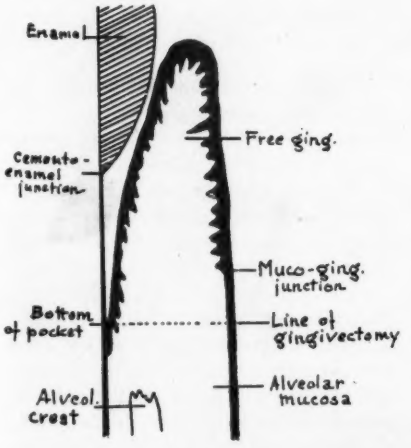

Fig. 3. Diagram, illustrating a periodontal pocket—the base of which reaches below the muco-gingival junction.

Fig. 4. A case in which the bottom of a periodontal pocket on the lower second bicuspid reached below the muco-gingival junction.
By a flap operation the gingival attachment was shifted apically to place the attached gingiva in an apical position.

along the margin for about two millimeters and the flap sutured—not in its original position, but farther apically. After healing, the surface appeared on a lower second bicuspid as shown in Figure 4. The attached gingiva was repositioned farther apically than it had been previously. The pocket was eliminated, but the new gingival margin was still formed by the attached gingiva. The inner surface of the attached gingiva was again attached to the tooth and the alveolar crest.

Several cases have been treated by this method and found satisfactory after more than two years of observation.

SUMMARY

A method is described to reposition the attached gingiva to the alveolar crest in cases where the base of the deep periodontal pocket was located apically to the muco-gingival junction.—*1014 S. Texas Bldg.*

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The Effect of Toothbrush Type on Gingival Health

by J. J. HINIKER,*D.D.S., and B. K. FORSCHER,** *Ph.D., Washington, D. C.*

THE importance of the toothbrush, correctly and frequently used, as a therapeutic agent in the control of periodontal disease has been emphasized by many workers. However the question remains as to the most desirable type of brush in regard to bristle hardness, design and bristle material. A number of different answers to this question are possible on theoretical grounds. For example, it is to be expected that the use of a harder brush should lead to increased keratinization of the gingival tissue. This has been confirmed experimentally by Stahl, et al. using histological techniques.[1]

This paper describes a pilot study designed to investigate the effects on the gingival health of various types of toothbrushes in a group of young adult males and to determine the feasibility of a large scale study of a similar nature.

EXPERIMENTAL

Materials—

Five types of brushes were studied. All brushes were supplied by a single manufacturer for purposes of uniformity. These brushes were:

1. Soft Natural Bristle 3 Row (18 knot)
2. Hard Nylon 3 Row (18 knot)
3. Hard Natural Bristle 3 Row (18 knot)
4. Soft Nylon Multi-Tufted (55 knot)
5. Soft Natural Bristle Multi-Tufted (55 knot).

Method—

A group of ninety young adult males from the First Year class of Georgetown University Dental School were examined and the condition of their gingival tissue was recorded by the P M A method of Massler, Schour and Chopra.[2] Only the eighteen anterior units in each arch were considered so that the worst score possible would be 36. All examinations were performed by the same clinician. The results of the first examination are shown graphically in Figure 1. At the time of examination, a color photograph was taken of each mouth.

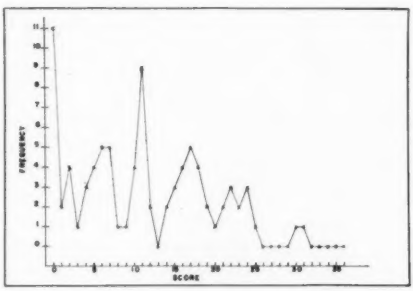

Fig. 1. Distribution of Original Scores.

The subjects were divided into five subgroups on the basis of the original examination so that the distribution of varying degrees of gingival health within each group was in the same proportion as in the sample as a whole. To each sub-group was assigned a specific type of brush and each subject received two identical brushes with directions to use them alternately. Instruction was given in the Modified Stillman-McCall Method[3] of brushing the teeth but no directions were given concerning frequency of brushing or type of dentifrice. It was desired to duplicate, in so far as possible, the normal oral hygiene habits of the group with the exception of method of brushing and type of brush.

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TABLE 1
Comparisons

Between Examination	Time Interval
1 and 2	First three weeks
2 and 3	Subsequent nine weeks
1 and 3	Overall twelve weeks

Examinations and photographs were repeated after a three week interval and again after an additional nine week interval. The examiner (JJH) was never aware of the previous score of the subject being examined nor of the type of brush in use. The results of each examination were recorded on cards, one for each subject for each examination, similar to those suggested by Massler et al.[2]

After the last series of clinical examinations had been completed the colored slides were considered to provide a second method of evaluation. The slides for each subject were projected onto a screen by a Vu-Graph for simultaneous examination and comparison. The set for each subject was graded in their relative order of health by two members of the Department of Periodontia (Drs. Hiniker and Paulsen). The slides were projected so that there was no relation between the sequence on the screen and the chronological order of the pictures. Further, the examiners had no knowledge of the previously recorded P M A scores.

It should be pointed out here that in a series of Kodachrome transparencies variations in color tones due to photographic technique will be found. However it was felt that comparison between pictures would be possible by judicious consideration of the relative color shades within each picture.

RESULTS

Precise mathematical comparison of results obtained with the different brushes was made difficult by the fact that, due to negligence on the part of some subjects and other extraneous factors, it was not possible to obtain a complete set of three scores for every subject. Rather than reduce the number of observations by discarding all incomplete sets, a mathematical method of comparison was devised to supply relatively equivalent values for the final consideration while still using all available data.

Instead of working with gross changes in total scores, the changes possible in the individual units of the P M A system were examined. Comparing one examination with a prior one, it will be found that an inflamed site will have returned to normal, an inflamed site will have remained inflamed or a normal site will have become inflamed. Therefore for each pair of examinations the data for each brush type were divided into three categories:

1. The total number of new units of inflammation.

2. The total number of units, previously inflamed, now returned to normal.

3. The total number of units remaining inflamed.

In order to account for the differences between numbers of subjects in different groups at different examinations, the final comparison was based on two ratios among the three categories. For each set of three sums, there were calculated:

a. $\dfrac{\textit{New units}}{\text{Previously inflamed units returned to normal}}$

b. $\dfrac{\textit{Units remaining inflamed}}{\text{Previously inflamed units returned to normal}}$

The general gingival health will be improved if a large number of units return to normal and a small number of new inflamed sites appear. Conversely a poorer gingival condition will be due to the ap-

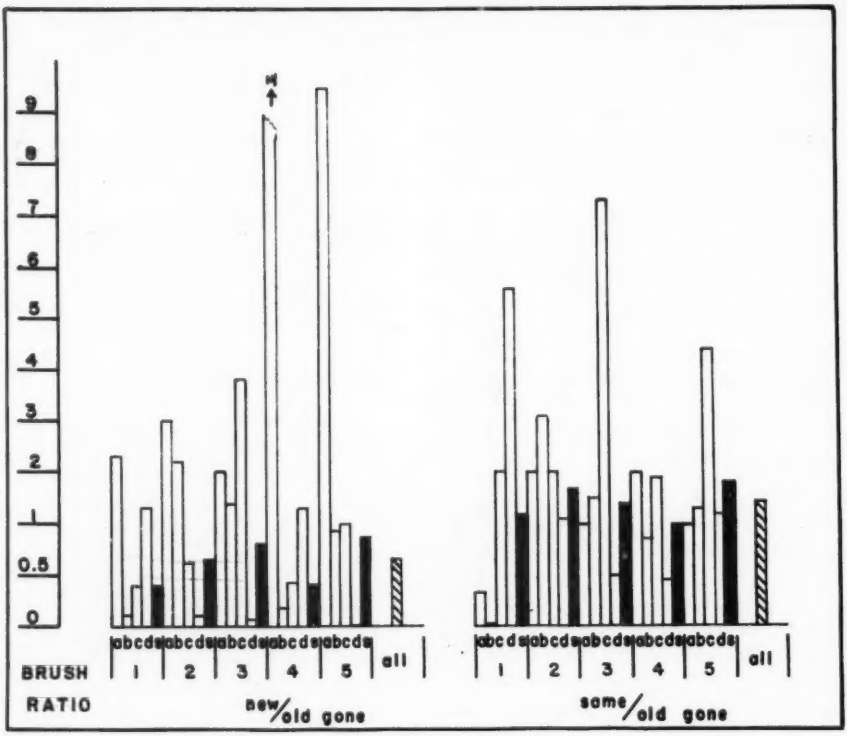

Fig. 2. Results as Bar Graphs, First Three Weeks.

pearance of a large number of new sites without disappearance of inflammation at the old ones. Therefore, in considering the overall change occurring between two successive examinations, high values for the ratios *a* and *b* indicate deterioration and low values indicate improvement. In comparing groups, the lower the values, the more desirable the change.

On the basis of the three examinations, three comparisons are possible as shown in Table 1.

The bar graphs shown in Figures 2, 3, and 4 present the data for the five brushes calculated according to the ratios described above. It will be noted that the values given for each brush group are divided into subclasses *a*, *b*, *c* and *d* as indicated by the open bars. The *a* designation represents the subjects with initial scores of 0 to 3; *b*, initial scores from 4 to 12; *c*, initial scores from 13 to 19; *d*, initial scores of 20 and over. In this way the effects of the specific brush type can be related to the original condition of the gingival tissue. The solid bar, designated *s*, represents the calculated ratio for the brush group taken as a whole without regard to original condition. To aid the comparisons, an additional calculation designated *all* and represented by the crosshatched bar is included which corresponds to the ratios obtained by using the sums for the entire sample without regard to original conditions or type of brush.

It is also possible to consider the results

TABLE 2

Results by P M A Scoring

Brush	First Three Weeks			Subsequent Nine Weeks			Overall Twelve Weeks		
	% I	% S	% W	% I	% S	% W	% I	% S	% W
1	70	12	18	20	27	53	62	31	7
2	33	13	54	55	0	45	50	8	42
3	37	19	44	60	13	27	53	13	34
4	77	0	23	33	8	59	60	20	20
5	44	0	56	43	21	36	50	7	43
overall	52	9	39	42	15	43	56	17	27

I, improved; S, remained same; W, became worse

in another manner. Again comparing the second examination to the first, the third to the second and the third to the first, it is possible on the basis of the total P M A score for each subject at each examination, to determine the numbers improving, remaining the same and becoming worse for each brush group. In order to account for the different group sizes at the different examinations, the numbers were recalculated to percentages which are given in Table 2.

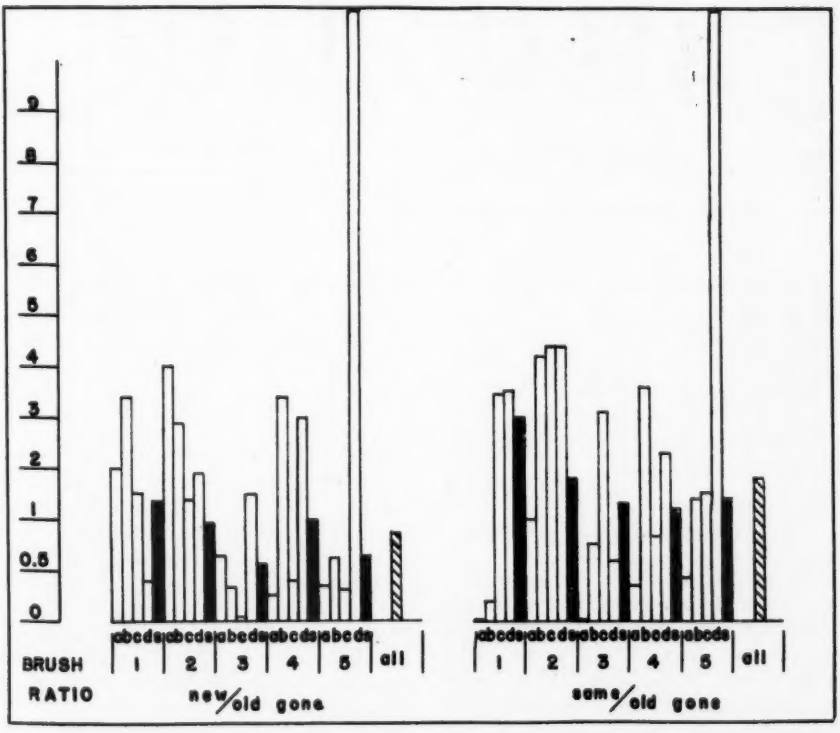

Fig. 3. Results as Bar Graphs, Subsequent Nine Weeks.

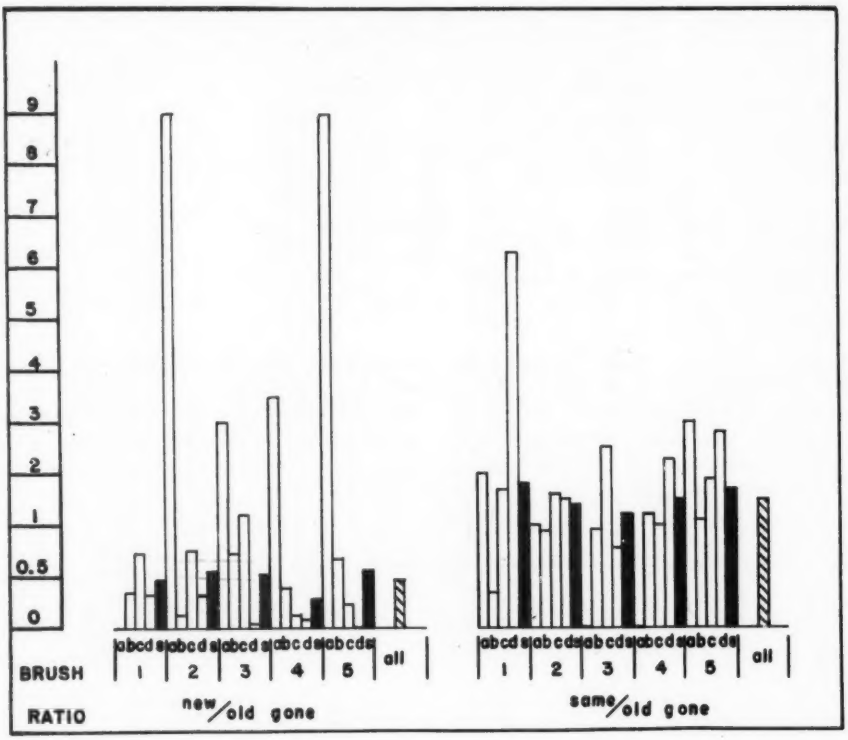

Fig. 4. Results as Bar Graphs, Overall Twelve Weeks.

The same type of comparison table can also be calculated on the basis of the ranks assigned by examination of the color transparencies. These results are given in Table 3. In considering this table it should be remembered that, except for one case, the category "same" was not used when the pictures were evaluated.

CONCLUSIONS AND DISCUSSION

The bar graphs shown in Figures 2, 3 and 4 should be interpreted by comparisons among bars rather than by consideration of any one bar alone. It will be seen that Brush 4, Soft Nylon Multi-Tufted, gives the shortest solid bar most frequently, and therefore on this basis is selected as the best brush from the point of view of gingival health, with Brush 1, Soft Natural Bristle 3 Row running a close second. However the differences between the results for all brushes are not striking.

The breakdown according to the original condition of the patients requires detailed study and does not lead to clear-cut conclusions in every case. It is of interest, however, to note how the responses differ. In the majority of cases a desirable response in the *a* and *b* sub-groups for any one brush for any one comparison is coupled with an undesirable response for the *c* and *d* sub-groups and vice versa. This confirms the theoretical expectation that no one brush type will be the perfect answer for all mouths.

The results as tabulated in Table 2 are

TABLE 3

Results by Inspection of Pictures

Brush	First Three Weeks			Subsequent Nine Weeks			Overall Twelve Weeks		
	% I	% S	% W	% I	% S	% W	% I	% S	% W
1	47	6	47	71	7	22	64	7	29
2	27	0	73	55	0	45	64	0	36
3	33	0	67	17	0	83	9	0	92
4	63	0	37	33	0	67	47	0	53
5	81	0	19	29	0	71	36	0	64
overall	51	1	48	40	1	59	43	1	56

I, improved; S, remained same; W, became worse

in general agreement with the bar graph representation. If the percentages for "improved" subjects are compared to the corresponding figure for the group as a whole, it will be noted that Brushes 4 and 1 make the best showing. According to Table 2 and to Figure 3, Brush 3, a Hard Natural 3 Row, shows the best improvement when the twelve week examination is compared to the three week examination, but the overall comparison, Figure 4, shows that in spite of the improvement during the second nine weeks of the experiment, Brush 4 still yields a better result.

Table 3 presents the results of the evaluation of the pictures tabulated in the same manner as Table 2 for the PMA scores. The use of color transparencies allowed simultaneous inspection of each mouth in its condition at each of the three examination times. In this way changes in the judgment of the clinician between examinations would be avoided. However accurate evaluation from slides is made difficult by the poor precision of color reproduction and by elimination of palpation as a diagnostic tool. There is poor agreement between Tables 2 and 3, necessitating a choice between ranking by PMA scores and ranking by inspection of color photographs.

Consideration of the original data cards on which the examination results were recorded leads to the choice of ranking by PMA score as the more accurate method, if consistency is taken as a criterion of precision. If a comparison is made among the three cards for each subject, it is noted that the pattern of inflamed sites is reasonably constant, indicating that the examiner maintained reasonably standard judgment throughout the series.

Although the presentation of results as in Table 2 is more simple than the complex ratio used in the calculation of the bar graphs of Figures 2, 3 and 4, it is felt that the latter method gives a truer picture. When results are classified simply as "improved", "remained same" or "became worse", large changes become equivalent in weight to small changes. For relatively small groups consideration of the actual changes in the involved units gives a more detailed summary than would be obtained from comparison of total scores. In order to evaluate the statistical significance of the observed differences, however, the original data, not the ratios, must be used. A statistical analysis is not presented here because of the small numbers in the brush groups. The groups were deliberately chosen to give a wide range of values and application of the standard methods of analysis would be misleading.

SUMMARY

1. The following brushes were tested for their effect on gingival health:

1. Soft Natural Bristle 3 Row (18 knot)
2. Hard Nylon 3 Row (18 knot)
3. Hard Natural Bristle 3 Row (18 knot)

4. Soft Nylon Multi-Tufted (55 knot)
5. Soft Natural Bristle Multi-Tufted (55 knot).

The test group using each brush contained individuals in various degrees of gingival health. There were 17 subjects in each group.

2. Results were recorded by P M A scoring for 36 anterior units and by color photography at time 0, 3 weeks and 12 weeks.

3. The results for the various brushes were very similar. However Brush 4, Soft Nylon Multi-Tufted, appeared slightly superior with Brush 1, Soft Natural Bristle 3 Row, a close second.

4. A method for comparing P M A scores for small groups of unequal size is suggested.

ACKNOWLEDGMENTS

The authors wish to thank Dr. A. G. Paulsen for his advice and assistance in this work.

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The Effects of Tensional Clenching Upon the Structures of the Neck

by IRA FRANKLIN ROSS,* D.D.S., *East Orange, New Jersey*

DURING the past few years, an increasing number of reports of the psychosomatic aspects of dentistry and medicine have appeared in the literature. As a result, there is better understanding of numerous problems, which previously had been complete mysteries because their behavior patterns did not conform to the classic laws of physiology and pathology.

Borland[1] recently stated that "frequently neuroses take the form of somatization of emotional conflicts and this is particularly true of the neuroses which are referred to as the 'hysterias.' " He went on to say that "the hysterical person is typically an immature one who finds it impossible to accept the responsibilities of adult society. The behavior, attitudes, and feelings are a frantic attempt to regain the position of dependence enjoyed by the person as a child. The impossibility of this in most cases, and its lack of satisfactory consequences when it can be accomplished, maintains an almost constant level of frustration and anxiety."

Thoma[2] and Stolzenberg[3] each reported instances of hysterical trismus. Moulton, Ewen, and Thieman[4] reviewed the literature extensively and discussed the direct and indirect routes by which emotional tensions may produce their results. Miller[5] stated that emotional tension may create oral disease through a disturbance of physiologic activity as seen in bruxism and in changes in salivary composition. In the investigation and treatment of these,

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Miller[5] quoted Campbell as saying that the one essential tool for exploration of this field (psychosomatic medicine) is good history taking." He stated further that the worries, fatigue, and frustrations of the patient are to be studied as well as the circumscribed disease process.

CASE REPORT

The case report which follows demonstrates some effects of tensional factors on the mouth and neighboring structures.

1. *Chief Complaint:*

The patient, a white female, aged 35, was first seen on January 27, 1953, at which time her chief complaint was recurrent, bilateral swellings and burning pain in the neck. She located these swellings along the anterior and posterior borders of the left and right sterno-cleido-mastoid muscles, and under the body of the mandible in the region of the right and left sub-maxillary glands. The patient also said that she occasionally had an itchy, dry feeling in her throat.

Further questioning about the cervical swellings revealed that they consisted of small, tender, bulging nodules which swelled and subsided every day, and that their distribution varied so that different nodes in the area became enlarged and tender on different days. Usually, chewing would cause a subsidence of the swelling and tenderness; but these enlargements would also frequently subside, sometimes quite rapidly, without the patient's resorting to chewing or to any other conscious mandibular movement. On the day of her first appointment, the patient called a few hours before the appointment time to say that the swellings were present at that time, and that she wanted to come right into the office so that they might be seen. However, during the half-hour necessary for her traveling, the swellings subsided completely, so that the nodes were neither seen nor palpable at examination.

2. *History of Present Illness:*

Three years earlier, at her post-natal check-up following the birth of her second child, the patient was asked by her obstetrician how long the glands of her neck had been swollen. Until that moment, the patient had not been aware of any swellings in her neck; however, the question caused some concern on her part and she decided to investigate further. About six months later, she consulted an otolaryngologist, who felt that a post-nasal discharge might have been responsible for the adenopathy. During his examination, he made the statement that there was nothing seriously wrong with the patient and that she should not worry about developing leukemia. Prior to this statement, the patient had not known that there might be a connection between leukemia and neck swelling. However, following this mention of the dread disease, she forgot his reassuring statement and remembered only that some forms of leukemia may be accompanied by a swelling in the neck. Also, two of her uncles had had cancer of the throat and she now began worrying about her swellings being caused by a malignancy. She next consulted an internist, then a surgeon, and then an endocrinologist. As a result, a growth was discovered on her thyroid gland, which after removal and microscopic examination proved to be a non-toxic colloid goiter.

About one and a half years later, the patient began noticing burning pain over both right and left sterno-cleido-mastoid muscles; and soon after, the swellings, previously described, appeared. After the onset of the burning she had several more consultations; all of which revealed no demonstrable pathology, clinically or roentgenographically, of the jaws, salivary glands, or neck.

The patient stated that she had become increasingly apprehensive following the onset of the swelling and the burning pain and frequently pressed her neck to palpate for nodes. She stated that this procedure frequently increased the pain, but she continued to follow it because she felt compelled to determine the presence or absence of swollen glands.

3. *Past Medical History:*

Consultation with the patient's physician revealed that her past medical history was negative except for nephrosis, which developed one year ago after the birth of her third child. Her first child was born eight years after her marriage; this delay having resulted from, first, unfavorable economic factors, and second, World War II. Unfortunately, this child was born with a congenital heart lesion and died soon after birth. When the patient was first seen by the author her two living children were aged three and one.

Questioning about her earlier life revealed that that she was the oldest of three children. She stated that she had been extremely lonely during her childhood and adolescence and therefore cried a great deal of the time. She felt rejected by her parents who were both actively engaged in business during her childhood and, as a result were unable to spend much time with their children. During the first year of her marriage her crying became much less frequent; but she did "flare up" (patient's description) occasionally if a disagreement arose between herself and her husband. However, because her husband suppressed his emotions, she felt that she also should learn to control her feelings. She did this and, as a result, felt that

she, her husband, and her children all lived together harmoniously because there were never any outward bursts of emotion or any violent disagreements. However, she went on to report, she frequently clenched her teeth during the day if she became emotionally upset. She also felt that she clenched her teeth during her sleep as evidenced by a tired sore feeling in the region of the temporomandibular joint when she awakened in the morning. In addition, she invariably clenched her teeth when picking up one of her children. Interestingly enough, she described this tensing action as a pleasurable sensation and felt that it was co-ordinated with the act of hugging the child. In as much as she enjoyed holding and caressing her children, she performed this clenching and hugging act many times during the day.

4. *Course of Treatment:*

At the initial visit, the history taking was begun, and the intra-oral clinical and roentgenologic examinations were carried out to determine whether chronic oral infection were present which might have accounted for the bizarre cervical swellings. Occlusal films were also taken to investigate for salivary calculi which might have been present but which had not shown up in the previous extra-oral or intra-oral films.

The patient returned one week later and the examination was completed. She was informed that no demonstrable pathology of the oral tissues was found. The clenching and palpating habits were then investigated and it was suggested that a harmless habit be substituted for the destructive tensional clenching.[6] This habit consisted of having the patient keep her lips together and teeth apart in a relaxed position during periods of emotional stress and while caressing her children.[6] She was also reassured that her condition was not serious and, in addition, was told not to continually press her neck, as this self palpation might be irritating and might be a factor itself in intensifying her neck symptoms. She returned one week later and stated that her neck felt greatly improved. Both the pain and the burning had disappeared although she though that a slight lymphadenopathy might still be present. On examination, no nodes were palpable. In addition, she stated that she missed some of the pleasure of holding her children when she did not clench her teeth. She substituted kissing the children for clenching.

One week later, she phoned to say that the pain and burning were completely absent. She also said that during that week she felt that she had regained the pleasure of holding her children without resorting to clenching her teeth.

The patient again called the office July 2, 1953, about five months after the initial visit, at which time she said that she still felt complete absence of pain and burning in her neck.

On October 19, 1953 the patient came into the office with the complaint that she had noted one area of swelling in her neck during the previous week. However, no pain had recurred. On examination no swelling could be seen or felt. However, to again allay the patient's apprehension a complete blood count and a heterophil agglutination test were suggested. The results were negative for any blood dyscrasias and infectious mononucleosis.

It was felt that this possible recurrence of neck swelling may have resulted from tensional clenching related to an anxiety state. Again, this swelling reported by the patient was neither seen nor felt by the examiner. The patient was again reassured that no pathology was present. She called a few days later to report that the neck swelling had not recurred.

COMMENTS

1. The initial cervical adenopathy, which was brought to the patient's attention during a post-natal check-up by her physician, may have been due to a permanent or transitory pathologic process in the area drained by the anterior and posterior chains of cervical lymphatics.

2. A statement by an otolaryngologist during a subsequent examination that the swellings were not related to leukemia actually initiated new fears.

3. Following her awareness of the problem, the patient aggravated the condition by constantly palpating the area.

4. As concern for herself increased, the patient clenched her teeth more frequently, both during the day and night, thereby causing increased muscular spasms leading to the subjective symptom of pain.

5. Subsequent adenopathy complained of by the patient, when first seen by the author, may have been real or imaginary, in as much as it was not present at the first appointment or during any subsequent appointment.

6. Complete remission of the pain and burning, which had been present for a year and a half, was produced by listening to the patient's story, explaining the mechanism of her problem to her, reassuring her that no serious disease was present, and

substituting a harmless habit for one capable of producing injury.

SUMMARY

1. A case of pain, burning, and possible adenopathy of the neck has been presented. It is felt that these symptoms may be related to chronic muscle spasms, resulting from frequent tensional clenching.

2. The patient's fears and apprehensions were greatly intensified because of misunderstanding and confusion resulting from numerous diagnostic interviews.

3. The patient responded immediately and favorably to consultative therapy and to the positive suggestion to replace a harmful and injurious habit with a harmless one.

4. A temporary recurrence of cervical swelling, eight and one half months after the initial visit, may have resulted from tensional clenching related to an anxiety state. Reassurance and favorable laboratory reports again brought relief.

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ARMED FORCES INSTITUTE OF PATHOLOGY OFFERS SPECIAL POST-GRADUATE COURSE IN ORAL PATHOLOGY TO BOTH DENTISTS AND PHYSICIANS

During the period March 8-12, 1954, a Post-Graduate Course in Oral Pathology will be presented at the Armed Forces Institute of Pathology, it was announced by Colonel Joseph L. Bernier, DC, USA, Chief of the Oral Pathology Section. Approximately 50 will be in attendance, of which one-half will be dentists and one-half physicians from both the Armed Forces as well as from the civilian professions.

The Faculty will consist of Doctors Isaac Schour, of the University of Illinois, Doctor Myron Aisenberg, of the University of Maryland, Doctor Lester R. Cahn, Columbia University, Dr. Helmut Zander, University of Minnesota, Doctor Joseph P. Weinmann, University of Illinois, and Doctor Harry Sicher, Loyola College of Dentistry, Chicago, and Colonel Joseph L. Bernier, DC, USA, of the Armed Forces Institute of Pathology.

This course is being given in response to numerous requests from both dentists and physicians and will include discussions on inflammatory, neoplastic and traumatic diseases of the oral regions.

This is the first attempt to bring instruction in pathology of the oral regions to members of both professions simultaneously. The quota for this course is completely filled, however, because of its popularity, it will probably be given again in the near future.

Those wishing further information should write to Colonel Joseph L. Bernier, DC, USA, Chief of the Oral Pathology Section, Armed Forces Institute of Pathology, the Course Director.

Case Reports

Extreme Occlusal Wear in a Young Adult

by PAUL GOLDHABER* *and* HYMAN GOLDBERG,** *Camp Chaffee, Arkansas*

IN a recent paper concerned with bruxism[1], a case report of a 51 year old farmer with extreme wear of the occlusal surfaces of all the teeth was presented. Because no positive history of night-grinding could be obtained and since the patient "had for a number of years farmed in Central Washington and had spent a great many hours and days driving a caterpillar type tractor," it was concluded that the occlusal wear was caused by riding a tractor. In view of these findings, the following case report may be of interest.

Fig. 1. Marked occlusal wear of mandibular teeth.

CASE REPORT

A 22 year old, white, married male, was admitted to the Hospital Dental Clinic, United States Army Station Hospital, Camp Chaffee, Arkansas, for routine dental treatment. At the time of the admittance, D. L. B. had been taking basic training for approximately 3 months.

GENERAL HISTORY

The patient was born in Freedom, Oklahoma, but has been residing in Alva, Oklahoma, for the past 21 years. He was one of four siblings (2 sisters and 1 brother), the youngest of which (male) died of leukemia during the past year. To the patient's knowledge, the other siblings, as well as his parents, were enjoying good health. The patient appeared in good health and temperament. His height was 6′1″ and his weight 175 pounds. Because of the recent history of leukemia in his immediate family, appropriate hematological studies were performed and the results were found to be within normal limits.

ORAL FINDINGS

Examination of the patient's mouth revealed a marked occlusal wear, particularly of the mandibular molars and premolars (Fig. 1). The lingual cusps of the maxillary and the buccal cusps of the mandibular teeth were completely worn down, exposing the dentine of practically every tooth (Fig. 2). The free-way space, how-

Fig. 2. Exposed dentine of mandibular teeth.

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Fig. 3. Protrusive position, showing inverted "V" wear-pattern of lower anteriors.

ever appeared to be normal. The uneven wear-pattern of the lower anterior teeth was of interest, since it simulated an inverted "V" (Fig. 3). This finding may possibly be explained by the marked horizontal overbite (Fig. 4), inasmuch as the lower anterior teeth could not always be in contact during the nonfunctional movements of the mandible.

Despite the obvious, severe occlusal trauma, the periodontium was healthy. Except for a slightly pale color and several anterior papillae which were missing or receded (Fig. 3), the gingivae appeared normal. There was no apparent mobility of the teeth, and full-mouth x-rays, including bite-wings, failed to reveal any bone loss.

PATIENT'S OCCUPATION AND HABITS

During the past ten years the patient worked on a farm, where his primary duty was driving a tractor. He claimed that this tractor-driving was usually performed for 16 hours per day during three months of the year, and 8 hours per day during two months of the year. The remaining months of the year were confined to other farming chores. A great amount of dust and grit was encountered during the plowing and cutting season. The patient was questioned about grinding habits. He stated that he had been unaware of this habit until he was told by his wife (married 8 months) that he grinds his teeth while sleeping.

Fig. 4. Marked horizontal overbite, showing impingement of lower incisors on palatal gingivae.

DISCUSSION

Comparison of the above case with the one previously reported in the literature reveals some interesting similarities and differences. The two cases resemble each other in that they both concerned farmers who drove tractors and showed extreme occlusal wear. A major difference, however, is the age factor, which probably explains the failure to find any periodontal breakdown in this case. While it is not surprising to find occlusal wear in a fifty-one year old patient (even in non-farming areas), extreme wear in a 22 year old patient is rarely observed. Unlike the older patient, however, a positive history of night-grinding was obtained from the younger patient. It should be noted that this positive history would have been overlooked if the patient had not been married. Therefore, in considering cases where night grinding is suspected, one should ascertain whether someone is available, other than the patient, to confirm whether the patient has been grinding.

With regard to this case, it seems reasonable to assume that the extreme occlusal wear was due to a combination of tractor-riding and night-grinding. At present, any attempt to evaluate the relative importance of these two factors in the etiology of the occlusal wear would be mere speculation.

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Alveolar Bone Resorption Associated with Diabetes

by HENRY M. SWENSON,* B.S., D.D.S., *Indianapolis, Ind.*

GLICKMAN[1] has shown in animals with experimental diabetes that non-specific pathological destructive bone changes occurred in a large number of diabetic animals while no comparable changes occurred in the control group. The changes observed were a loss of tooth-supporting bone independent of any alteration in the gingival crevice. He concluded that incidence of periodontoclasia would be greater in diabetics than in normal individuals.

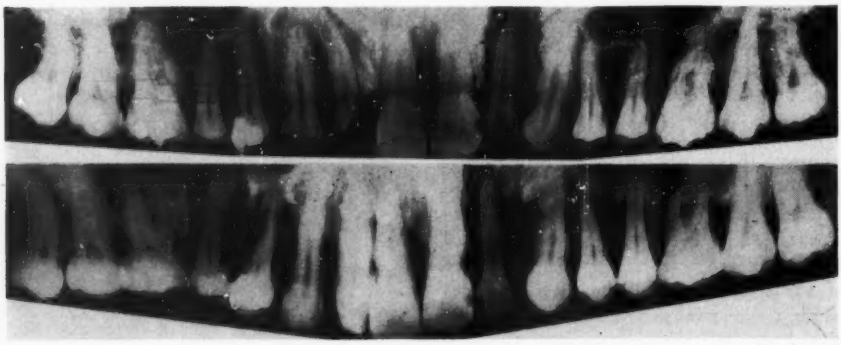

Fig. 1. Panoramic view of Radiographs showing extensive bone resorption in a patient with uncontrolled diabetes. The lower radiographs were taken thirty-five months after the upper ones. Radiographs of upper arch.

The case to be presented here substantiates this point of view since it involves an excessive amount of alveolar bone loss which occurred after the onset of diabetes. The patient is a 28-year-old unmarried male who was first diagnosed as a diabetic in September, 1945. He was placed on a restricted diet which was not adhered to. Most of his meals were eaten in restaurants and therefore it was difficult to follow dietary recommendations and he soon gave up entirely. In March, 1948, he went to his dentist (Dr. T. Esmon) for a prophylaxis. At that time a mild gingivitis was present which responded fairly well to treatment. The full-mouth x-ray showed a slight disturbance in some alveolar crests. The patient did not return for treatment again until February, 1951, when he stated that his lower left incisor had fallen out by itself.

Clinical examination showed marked mobility of the teeth. The gingival tissue was inflamed and hyperplastic with suppuration present around most of the teeth. X-ray examination revealed extensive bone loss around all of the teeth.

On questioning the patient, it was found he was not following his diet and was not taking insulin. He was referred to his physician who placed him on insulin and impressed on him the importance of proper diet. He returned to the dental office for treatment of his periodontal condition. When the periodontal symptoms had been

*Periodontia Department, Indiana University, School of Dentistry.

1. Glickman, Irving, "The Periodontal Structures in Experimental Diabetes," New York Journal of Dentistry, **26**:226, June, 1946.

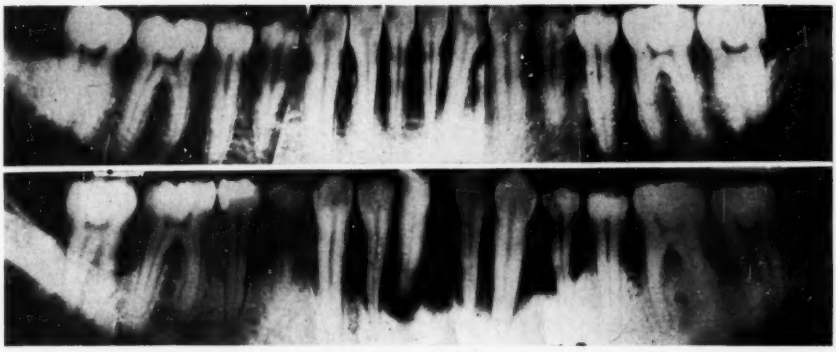

Fig. 2. Radiographs of lower arch of same patient, taken thirty-five months apart.

reduced, the teeth were extracted one or two at a time and they healed without incident.

This case illustrates a dramatic bone loss in a young individual with uncontrolled diabetes. It is difficult to postulate why this occurs in some diabetics, and many possibilities have been offered. It is not within the scope of this care report to discuss the various mechanisms involved in bringing about this condition, but the following is suggested: The uncontrolled diabetic is usually in a negative nitrogen balance which results in drainage of the body proteins. Adequate protein metabolism is necessary for calcium metabolism. Therefore, it might be possible with a reduction in proteins to have a disturbance between bone formation and bone resorption.

Regardless of the mechanism involved, it does emphasize the relationship between *some* systemic disturbances and *some* periodontal involvements.

The Use of Vitamin "A" in the Treatment of Periodontosis

by T. A. LEACH, D.D.S., *Hutchinson, Kansas*

MUCH has been written and various theories advanced as to the cause and cure of periodontia, or periodontoclasia, but the conclusive facts have, so far, been largely negative. Many have observed mouths in which the teeth have been beautifully arranged, with normal occlusion, and years later were lost because of periodontal infection. We also have mouths with irregularly arranged teeth with poor occlusion, which were not lost because of periodontosis or traumatic occlusion. One often observes the teeth of the individual who has acquired the habit of masticating on one side, and notes the teeth on the masticating side are firm and the gingival tissues are in a state of health, while on the opposite side the teeth may be slightly loose, the gingival tissues congested and the teeth more or less sensitive to masticating pressure.

In the not far distant future we may

discover that the diet and exercise plays an important influence in the pathologic condition of the periodontal tissues. Wide varieties of food do not require enough mastication to give the teeth and supporting tissues sufficient exercise. A deficiency in the food elements which contain vitamin A appears to exert a deleterious effect on the periodontal tissues.

The hearing of the author was becoming progressively more difficult; therefore, the parenteral administration of 50,000 units of vitamin A concentrate was prescribed. This treatment semiweekly, was carried on over a period of four weeks, during which the hearing improved, but the most noticeable effect was the improvement in the condition of periodontosis which had been affecting the posterior teeth on the right side. For some time the teeth on that side had been somewhat tender to mastication and were slightly loosened. After the administration of the vitamin A concentrate the teeth tightened and all tenderness disappeared. That was two and a half years ago and the teeth have remained normal. However the diet since then contains foods rich in the vitamin A.

After the above personal experience the vitamin A concentrate in 50,000 units was used parenterally for two patients who had quite advanced periodontosis. The results were most gratifying to both patients and doctor. After three parenteral administrations of the vitamin A concentrate (Lobel formula, which is 50,000 units in ampules under the trade name of Anatola put out by Parke, Davis & Co.) the gingival tissues became normal in color, tenderness disappeared and all formation of pus around the teeth ceased. The teeth were scaled before treatment. The use of vitamin A food was suggested. The mouths of these patients have remained healthy over a period of more than two years.

During the last two years and over it has been routine practice to administer the Anatola parenterally in the treatment of periodontosis after scaling the teeth and in every case the results have been most gratifying. We have used the Anatola without scaling the teeth and the formation of pus ceased, but later were scaled to prevent a recurrence. One case was especially advanced, with very little chance for even fair results, but the results were amazing and the mouth is now in a healthy condition.

A Critical Review of "Psychosomatics and Suggestion Therapy in Dentistry" by Jacob Stolzenberg

by LOREN R. BORLAND, B.S., D.D.S.,* *San Francisco, Calif.*

IN the last decade the dental profession has felt the impact of a challenge stemming from its decreasingly naive patients on one hand and from some of its own more forward-looking members on the other—a challenge to face the responsibility for the welfare of patients rather than mouths. This has led to enthusiastic outbreaks of interest in psychology and the advocation of the use of hypnosis as a part of the routine dental armamentarium. It has also led to the publication of several books and articles dealing with dental psychosomatics and hypnosis in den-

*Philosophical Library, New York, 1950.

*Lecturer in Periodontology, University of California, San Francisco; Clinical Psychologist, Langley Porter Mental Hygiene Clinic, San Francisco.

tistry, and more publications on these and allied topics can be expected in the future. Such a trend is commendable, indeed, but it gives rise to a new and important responsibility—that of critically analyzing these publications and of demanding that they satisfy the same criteria of scientific rigor and objectivity which we impose on publications dealing with such subjects as caries control and etiology, fluoridation studies, etc. In our eagerness to accept this new and important extension of our domain we must strive to prevent the promulgation of half-truths, distortions, and rationalistic philosophy under the guise of "scientific truth." These trends make the critical evaluation of such books as Dr. Stolzenberg's imperative, and it is within this framework of values that this review is offered.

The discussions of the essential totality of the organism as presented by Dr. Stolzenberg are quite conventional and agree quite closely with the views of contemporary psychologists and philosophers of science. He stresses the ultimate futility of attempting to separate the *soma* from the *psyche* and illustrates his point with anecdotal remarks as well as various statistical analyses of psychosomatic studies, more or less classical in the field. He shows his bias, however, on page 47, where, in support of his thesis that emotional disturbances are important etiological factors in periodontal pathology, he quotes Dr. Edward J. Ryan as saying that "emotional stress may be a contributory factor in caries and periodontal disturbances by means of changing the chemistry of the saliva. . . ." Dr. Ryan offered this possibility in his book, *Psychobiological Foundations in Dentistry*, not as a conclusion or evaluation as a logical hypothesis which seemed worthwhile testing. He has indicated in conversations with this reviewer that such formulations were intended to point the way in a new area where virtually nothing was known empirically—that he would strongly emphasize the word *may* in the quotation. Thus Dr. Stolzenberg appeals to Dr. Ryan as a supporting authority for his contention of psychogenicity of periodontal disease—a relationship which Ryan had suggested as a *possibility* worthy of investigation. He seems to ignore the very basis of scientific method—that science admits of no such thing as a *fact*, but rather deals in *inferences* and *hypotheses* having various degrees of objective verification. The objective or factual status even of such a thing as the length of an object or the wavelength of a given light source is relative. Measurements by independent observers would very likely appear to differ to some extent if the measurements were carried to a fine enough degree. How much more difficult it is to maintain an objective attitude in the biological sciences is well known to all of us. We must not, however, assign objectivity a less important role in our work. This tendency for observations or statements to pass from the status of hypothesis to "fact" by the mere passage of time and the quoting of authority (frequently with no effort to maintain the original intent of the statement) is especially to be guarded against. Nothing in the way of empirical objective evidence is offered in support of any of the dental psychosomatic relationships put forth in the book, though they are all presented in a way which implies that they are substantial, objectively determined "facts."

Dr. Stolzenberg's treatment of neurosis and anxiety is a weird example of theoretical eclecticism and misinformation. He appears at the outset to pursue a rather psychoanalytically oriented course, but this yields to a mixture of homely philosophy and mysticism in which he suggests telling the patient to "think positive thoughts" and to "listen to the 'Unity Radio Broadcast' " as practical ways of handling problems which, from the case material presented, are obviously deeply ingrained neurotic processes. He attempts to utilize the psychoanalytical scheme of psychosexual development, explains it in a very misleading manner, and then moves on with only an occasional perfunctory and

uninformative reference to it.

The matter of the interaction between the dentist and his patient is suggested by Dr. Stolzenberg as a very important element in the practice of dentistry. This seems to be a safe assumption and one which gives rise to many hypotheses which could be tested empirically and perhaps verified. He offers it as a bald "fact," however, and proceeds to preface his statements with such propagandistic clinchers as "the melancholy truth is . . .," etc. This reviewer objects strongly to the use of the word "truth" in this context. Dr. Stolzenberg says further "It has been demonstrated during the recent war that the magnificent ability of the peoples of the Pacific Islands to withstand the most ghastly and intolerable situations of war without emotional crack-up—when American soldiers did not —was largely due to the unshakeable security these peoples learned by prolonged feeding at the breast in infancy, combined with no forcible toilet-training until the child itself undertook such training, and no prohibition on sexual play." It is true that the last war did demonstrate that many peoples of the Pacific Islands could withstand emotional trauma with less severe effects than could many American soldiers. However it is *inferred*, not demonstrated, that this greater resilience was due to the factors mentioned.

The fears and anxieties of dental patients and their precipitating factors are discussed very meaningfully and provocatively, with numerous examples of situations contributing to the persistence as well as the origin of such feelings. Dr. Stolzenberg next considers the proper role of the dentist relative to his patients' emotional characteristics. He waves a final adieu to a rapidly vanishing objectivity as he says that personality theories are too academic and impractical to offer a solution to the problem and suggests that the dentist resign himself to a metaphysical approach, adopting a "philosophy of behavior and a specific attitude regarding his patient." The obvious answer, says Dr. Stolzenberg, is for the dentist simply to develop an "emotionally mature" personality himself, and this will qualify him to deal intuitively with the complex problems confronting him. Thus the author has arrived, rather laboriously it would seem, at precisely the point at which the dental profession has found itself for decades—the position which says that the handling of patients as personalities is too complex for a systematic approach and must be undertaken on the dusty basis of homespun philosophy and intuition. Even if such a doctrine seemed justified—a point which this reviewer cannot accept—Dr. Stolzenberg would still be faced with the question of just how a dentist goes about sidestepping all the hurdles which impede the efforts of the country's best psychotherapists and proceeds blithely under his own power to a condition of emotional maturity. The attempt to correct or modify behavior patterns and personality characteristics by the simple expedient of giving good advice to be followed by the patient was quite uniformly discarded by psychiatry as early as the turn of the century.

The historical treatment of hypnosis is substantially accurate and interestingly and informatively written. The rather detailed instructions for the induction of hypnotic states and the use of suggestion are clear and easily comprehended. The main criticism this reviewer offers with respect to the treatment of hypnosis is concerned with the over-optimism Dr. Stolzenberg displays with regard to the results to be expected from hypnosis and the over-application of hypnotic measures which he advocates. He suggests that the dentist can, through the use of hypnosis and suggestion, effect minor therapeutic changes in his patients which will make of them better dental patients as well as helping them to achieve better adjustment in their daily lives. This appears to be a laudable pursuit but one not likely to be accomplished by the wholesale application of symptomatic treatment such as hypnosis. Dr. Stolzenberg says himself that "Suggestion does not concern itself with conflicts; it disregards the dynamics

or the motives of emotional states. Suggestion does not recognize conflicts, their cause and workings, nor how to handle them." This would certainly be supported by the vast majority of psychiatrists and psychologists today. It is difficult to understand how the author can follow such a statement as this with the advocation of treating neurotic fears, anxieties, phobic reactions, compulsive habits, etc., by the simple process of suggesting them out of existence. Such a naive approach to the solution of emotional problems and neurotic behavior bespeaks either a complete ignorance of decades of research in psychotherapy and personality theory, or an equally complete disregard for such a body of evidence. It would seem fitting that we as dentists should assume our share of the responsibilities in the psychological area, but we should be very cautious about walking into the field with very little knowledge of the subject and immediately proposing to revolutionize a rather respectable and well established scientific discipline.

Dr. Stolzenberg insists, as indeed every advocate of hypnosis must insist, that hypnosis is virtually incapable of harmful effects. This agrees with the opinions of most of the authorities who have contributed to the research in this area, but it is difficult to reconcile with the author's advice that the patient should be told, while in the hypnotic trance, that he will never submit to hypnosis except for medical or dental treatment. This would seem to suggest that either (a) Dr. Stolzenberg is not fully convinced of the harmlessness of the procedure, or (b) he is engaging in the same professional jealousy of which he accuses the psychiatrists.

The book is filled with contradictory statements. The reader is told not to use the word *impression* in speaking to a patient, but the author suggests on another page that the dentist introduce the procedure by saying, "I'm going to take an impression of your teeth today." Such conflicting statements are confusing, but not so deserving of criticism as are the many glaring errors in psychological theory—errors which might well escape the sincere and interested but psychologically untrained dentist. We are told "Anxiety and fear are the same. . . ." "Experiences which take place in the first five years are recorded in the subconscious mind by impulses. . . . The psychiatric term for the mental registration is referred to as a repression," and many other equally misleading statements. In many places it becomes quite difficult to decide whether a given error is due to original misinformation or to the rhetoric.

This reviewer sympathizes with any effort to acquaint the dental profession with what psychology has to offer. In that sense he appreciates the book and the intentions of the author. He feels, however, that the self-imposition of extremely severe standards of objectivity, empiricism, and scientific rigor is of basic importance in this area if it is not to lose its value. It is a new and precocious child and can easily arouse the ire and disdain of the dental profession if its infant pronouncements are too loud and irresponsible. For these reasons this reviewer cannot avoid voicing the opinion that *"Psychosomatics and Suggestion Therapy in Dentistry"* is less likely to result in a better understanding of dental psychology than it is to lead to confusion, disappointment, and general misunderstanding.

WESTERN SOCIETY OF PERIODONTOLOGY AND CALIFORNIA ACADEMY TO HOLD JOINT MEETING

There will be a joint meeting of the California Academy of Periodontology and the Western Society of Periodontology in Las Vegas, New Mexico on May 21, 22 and 23. This meeting will be held in the Hotel El Rancho Vegas and promises to be excellent. Anyone desiring further information may write Dr. Perry A. Ratcliff, President, Western Society of Periodontology, 1998 "D" Street, San Bernardino, California.

Report of the President of the American Academy of Periodontology 1952 - 1953

by ROY O. ELAM, D.D.S., *Nashville, Tennessee*

IT is appropriate that the American Academy of Periodontology meet during the last weeks of September, because everyone thinks in terms of returning to school in the early fall of the year. Since the object of this Academy is, as stated in its Constitution (Article II), "To advance the art and science of periodontology and by its application, maintain and improve the health of the public," all who attend this meeting are in a sense returning to school. It is our hope you will find this meeting so instructive —and enjoyable—that you will wish to return annually.

It is also appropriate that the Academy meet in Cleveland, Ohio, because it was in Cleveland on February 21, 1914, that a small group of periodontists wrote the series of resolutions which declared their intention of forming a society of those especially interested to "meet and work together without prejudice for the scientific investigation of dental caries and periodontoclasia"[1] and who believed "that the practice of Oral Prophylaxis and Periodontia as an exclusive specialty may be encouraged; and that the general profession may be made fully acquainted with the results that can now be obtained."[1] It is now well known that the Organization Committee,[2] meeting in May 1914, again in Cleveland, did set into motion the organization labelled The Academy of Oral Prophylaxis and Periodontology. The first meeting of this young academy was held November 5, 6, 7, 1914 and seventeen members attended. What a change has occurred in the intervening 39 years! According to estimates, this meeting more members and guests will be registered than ever before.

Far more important than the increase in attendance at meetings is the increased influence the Academy has on dentistry since its organization.

As retiring President, I wish to comment briefly on the various spheres of influence of the Academy.

I

THE ACADEMY

The American Academy of Periodontology is today recognized as a stable, well established organization, officially representing the specialty of periodontology. Through its influence periodontology has become accepted as an integral part of general dentistry, and an activity worthy of specialization. Most of the important work of the Association is carried on by committees and by the Executive Council of the Academy, and the Academy owes a debt to the many members who have served.

This is the first year the Academy has operated under the new and completely rewritten Constitution and By-laws. Changes over the years made it desirable to change many features of this Academy, including the method of selection of members. Since your present President was active in rewriting these new by-laws, he knows they are

1. From the preamble of the original Constitution of the Academy.

2. Composed of Grace Rogers Spalding, Detroit, Mich., Gillette Hayden, Columbus, Ohio, Clyde M. Gerhart, Washington, D. C., J. W. Jungman, Cleveland Ohio, J. Herbert Hood, Cleveland, Ohio, A. C. Hamm, Denver, Colo., and John Oppie McCall, Buffalo, N. Y., Chairman.

not perfect and will have to be modified occasionally to meet new demands.

Until the new Constitution and By-laws were adopted in St. Louis last year all of the standing committees were committees of the Executive Council. The new By-Laws establishes seven standing committees of the General Assembly who will report directly to the General Assembly and three standing committees of the Executive Council. They are all listed in the By-Laws.

Probably the most important committee is the new committee on Scientific Sessions composed of B. O. A. Thomas, Donald A. Kerr, and Robert G. Kesel as Chairman. This committee function is a new idea under which we are operating for the first time at this meeting. This committee has the full responsibility of all the Scientific programs of the Academy. Under this committee are three sub-committees, the program committee, the clinic committee and the local arrangements committee which they nominate to the Executive Council for election. They also have the privilege of nominating any special committee they may need.

Under the old By-Laws the members of committees were elected for a term of one year. Under the new, each member is elected for a term of three years with one member's term expiring each year. With this system there will always be two members of a committee with past experience and familiar with the work of the committee. It will be interesting to see how this system works and you will experience its first operation at this meeting. Since this is its first performance, do not expect it to work perfectly.

One of the contributions the Academy makes to organized dentistry is, of course, this annual meeting. I wish to express sincere thanks to the chairman of the Program Committee, Dr. Frank E. Beube, Chairman of the Clinic Committee, Dr. Everett A. Archer and Chairman of the Local Arrangements Committee, Dr. Marion L. Drake for their assistance, and especially are we indebted to the Committee on Scientific Sessions, Drs. Kesel, Kerr and Thomas. I know everyone connected with it has a feeling of gratification for having had a part in the planning of it.

The influence of the Academy has been stabilized by the conscientious steady work of the Secretary, Dr. Clarke Chamberlain and the Treasurer, Dr. Ray Johnston. Working with them is a pleasure and I wish to acknowledge their help. The knowledge that a task is well done is ample reward—in fact the only reward—for men who are conscientiously interested in serving the Academy, and certainly these men deserve to be pleased with their work.

II

JOURNAL OF PERIODONTOLOGY

The influence of the Journal of Periodontology continues to expand and through it the American Academy of Periodontology is exerting a healthy influence on the dental profession. On behalf of the Academy I wish to give the editor and his staff a vote of thanks for their services to the Academy and to dentistry. I am informed that the Journal now has the widest circulation it has ever had; certainly both the quality and quantity of its contents continue to improve. Please continue to give the editorial staff your help.

III

ANNUAL SEMINAR

It has become an annual custom for the American Academy of Periodontology to hold an annual seminar in Ann Arbor, Michigan. Dr. Grace Rogers Spalding initiated this

custom, and always was active in supporting it. This seminar usually deals with the relationship of developments in the basic sciences to periodontology and is worthy of the attention of everyone interested in periodontology. One need not be a member of the Academy to attend; the next session is to be in March 1954. As is always true, there are a few members who have been most active in organizing this seminar, and for the Academy I wish to thank Drs. Harry Wood, Edgar James and Joe Barkley for planning this important annual program, and Drs. Donald Kerr, William Mann and Paul Jeserich for making it possible to hold these seminars in Ann Arbor.

IV

Another sphere of influence of the Academy is through the activities of its individual members, by giving clinics and demonstrations, writing papers and reading papers before dental societies. It is gratifying to note that almost every large dental program includes a presentation on periodontology by some member of this Academy. This trend is one that we wish to see continued and so we earnestly request every member to take it upon himself to see that periodontology is given ample time on all programs for dentists. In this way we can make a definite contribution toward increasing the effectiveness of treatment of periodontal disease.

V

AMERICAN BOARD OF PERIODONTOLOGY

Another activity sponsored by the Academy of Periodontology is the American Board of Periodontology, which has the increasingly difficult task of certifying its diplomates. The Academy and the profession owe the Board members, particularly, of course, its Secretary, Dr. Harold Leonard, much for their services. This year for the first time a practical examination was included in their testing. Facilities of Indiana University were used.

In conclusion, I wish to thank the members of the Academy for their confidence in electing me President. I belong to many dental organizations and have served dentistry in many ways but I can say with sincerity that the Presidency of the American Academy of Periodontology has given me more personal pleasure and gratification than any other, and so I thank you for the honor and privilege of serving as your President and I hope I have fulfilled the obligations every President should assume. I believe the influence of the Academy has increased this past year, and it is my desire that it will continue to prosper. Recently Sir Richard Livingstone wrote that the prior task of education "is to inspire, and to give a sense of values, and the power of distinguishing what is first rate from what is not." I envision the Academy as an educational organization, performing these functions, and I trust its spheres of influence will broaden and deepen continually.

REPORT OF THE COMMITTEE ON THE PRESIDENT'S ADDRESS

The Committee to report on Doctor Elam's Presidential Address has met and studied the paper carefully. We wish to compliment him upon his thoughtful address.

The report contained no special recommendations, because of the fact that the report is a recitation of many accomplished recommendations previously made by Dr. Elam. As is well known, Dr. Elam was Chairman of the committee which completely revised the Constitution and By-Laws, and has been most active in bringing

about the adoption and application of them.

The success of this meeting and the increased activity of the other various standing committees gives evidence of the value of Dr. Elam's contributions. We are confident that the influence of the Academy will continue to expand under the organization now developed.

The Committee concurs in Dr. Elam's suggestion that each individual Academy member assume the responsibility of extending the Academy's influence through his efforts as a clinician and essayist.

The Committee is also pleased to reiterate the debt the Academy owes to its officers, and add to Dr. Elam's list his own name.

Respectfully submitted:
James C. Westbrook, Jr.
Allison G. James
Maynard K. Hine, Chairman

Résumé of Papers Presented at Meeting of American Academy of Periodontology, Cleveland 1953

Prepared by HAROLD RAY,* D.D.S., M.S., *San Francisco, California*

The thirty-seventh meeting of the American Academy of Periodontology was an excellent one and the material presented was worthy of dissemination. The following abstracts, prepared for local distribution, interested the Editor, who requested permission to publish them. They must be looked upon as "on the scene reporting" which is subject to possible error and misinterpretation by the writer. An honest attempt, however, has been made to report the actual papers, realizing some of the attendant difficulties of a large audience, distractions and so on. Many of the papers presented will be published in the Journal of Periodontology. Comments, labeled as such, in any individual paper are the writer's own opinion and not those of the author of the paper reported; they are injected as clarification and addition to stimulate individual thought along the provocative ideas that some of the papers may produce.

NORMAL PHYSIOLOGY OF THE PERIODONTIUM AS IT IS AFFECTED BY OCCLUSAL FUNCTION

by CHARLES WILLIAMS, D.D.S., B.S., *Toronto, Canada*

This paper was primarily a review of the material that Dr. Williams obtained in his examination of Eskimo dentition and was directed to one phase of the problem of physiology of the periodontium, that of occlusal wear or abrasion. He states that one of the premises that was worthy of consideration was the clinical validity of the ideas of passive and active eruption in relation to occlusal wear.

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*Associate Clinical Professor of Periodontology and Chairman of the Division, University of California College of Dentistry.

In correlation with his clinical observations of the Eskimos Dr. Williams also conducted some feeding experiments on animals and examination of some dry human skulls. Some of the general considerations, in his Eskimo work, were that the arches were well arranged and developed, and that the number of teeth present was very high. He felt that he could definitely say that no caries were present and there was a very little amount of periodontal disease and that which did occur was of the Simplex type. In three of the individuals that were examined he found the periodontium damaged as a result of wear. This judgment was from a total number of 3,400 patients. He showed slides of patients with considerable amount of occlusal wear but pointed out that the gingival margin of the gingiva was continuous at the cemento-enamel junction and therefore concluded clinically that the process of continuous eruption was not evident.

Dr. Williams pointed out too that in these people the occlusal wear, which he referred to as physiologic, eliminated in many instances any tendency toward overbite and illustrated the fact that as teeth wear the incisors approach more and more an end to end relationship. In substantiation of his finding that very few teeth were lost, he cited a table showing that at thirty years of age the patients had an average of 30.8 teeth; between 30 and 40 patients had an average of 30.4 teeth and from 50 to 60 patients had an average of 29.3 teeth. This of course is a high percentage of teeth remaining in any mass study. In an analysis of overbite he showed that in children up to ten years, 48% of them had no overbite; in individuals from 11 to 20 years of age 58% had no overbite and from 21 on up 87% of those patients had no overbite, substantiating the concept that wear eliminates overbite. The animal feeding experiments were done on rats and they were divided into two groups and one group was fed a hard coarse diet, which was gritty in some respects, and the other group was fed the same diet except that it was finely ground almost into a powder. The two groups were fed their respective diets for a period of four months and the group that had the coarse diet showed occlusal wear and the ones fed the soft diet did not.

Back to a consideration in humans he feels that gingival recession when it occurs is due to the very thin covering of the periodontium, particularly on the buccal and labial aspects, and this thin covering is due to inadequate development and inadequate function, stemming from inadequate physical properties of the diet. With a thick covering there is resistance to recession and it is not seen. Obviously the Eskimos live on a diet much different from ours and it has the physical properties of stimulating functional response. Dr. Williams also pointed out that a soft diet is conducive to bacterial growth and the resultant process of inflammation in gingivitis.

He expressed doubt concerning the ideas of continuous eruption and said he found no clinical evidence of such eruption. In his work on dry skulls he found that the facial measurements in his opinion were not disturbed as the result of wear. Dr. Williams pointed out that there were three possibilities that could occur in occlusal wear (1) was that the position of the tooth would be maintained (2) was that eruption perceptibly out of the socket as an expression of active eruption would occur and (3) eruption could occur and be followed by the apposition of alveolar bone at the alveolar crest as well as at the fundus, meaning of course that the tooth and the periodontium erupt together. Dr. Williams showed diagrams of what he terms four degrees of occlusal wear. Degree No. 1 was discernible wear in the enamel along the incline planes of the cusps. Degree No. 2 a continuation of the wear to the point where dentin is revealed. Degree No. 3 continuation of wear to the point that the cusps were missing and the dentin appears cupped. Degree No. 4 that of one half or more of the clinical crown being worn.

In measuring the dry skulls, Dr. Williams measured from the nasion to the point of the chin called the gnathion and from these points to mid-line of the maxillary alveolar ridge and to the mid-line of the mandibular ridge. He also made measurements from the ridge to the cemento-enamel junction. In the four degrees of wear only in the fourth degree did he find the face to be closed and in the fourth degree of wear he found definite differences in the height of the alvolar ridge and he found little difference in the measurement from the ridge to the cemento-enamel junction. In some patients the face was reduced five millimeters of facial height in the fourth degree of wear and the occlusal plane was not maintained. He maintains also that wear is a normal physiologic function and favorable to the periodontium. It produces an even distribution of loading and as aging occurs the two lever arms, that of the visible one and that in the periodontium are more nearly equalized, coincidentally the sharp inclined planes are reduced. He reiterated that as cusps during occlusal wear disappear inverted cusps appear. His conclusions were that Eskimos in general had no overbite and that an average of three fourths of a millimeter decrease in facial height was discernible in the four degrees of wear.

DISCUSSION

Dr. John Pritchard of Baylor University discussed this paper and pointed out that as wear occurred there was an increase in the buccal lingual diameter occlusally, in the teeth of the hard diet animals, and that a clinical consideration of this feature must be that in our occlusal equilibration we must reduce the extent of this diameter. He pointed out that Brodie has said that the position or the functioning of the teeth have no influence on the pattern of bone growth and he wonders whether or not an end to end bite is really the result of function. Dr. Pritchard also mentioned that high cusps and unworn incline planes are signs of immaturity and that a soft diet continues this immaturity. He also pointed out that balance in lateral excursion is not important except where the mandible becomes locked in occlusion and that the purpose of equilibration artificially must be to unlock the mandible. He also suggested that Jankleson's work shows the teeth do not come in contact in chewing and wonders why a good diet is indicated to produce physiologic wear.

Balint Orban discussed the paper and pointed out from Williams' own histologic material in the animals fed the two kinds of diet, that passive eruption was going on as evidenced by proliferation of the epithelial attachment apically. The epithelium seems to start its migratory course early and in the slides shown it was already apical of the enamel. He also reiterated that the ideal for passive eruption is that it would go slowly. He also pointed out that alveolar bone grows continuously along the alveolar crest and the fundus and this can be clearly seen in microscopic sections of both animal and human material and he said that there was a great difficulty in comparing clinical judgments with microscopic ones. To this Williams agreed.

Irving Glickman of Tufts pointed out that in studies that he had made on animals, a feature with which we are all familiar, was that *in animals* when the antagonists were removed in the upper, the lower teeth erupted with the periodontium following them.

COMMENTS

This is a very interesting study from which a few conclusions can be drawn. Perhaps one conclusion that is outstanding is that the periodontium is not necessarily injured by occlusal wear but to say that as a result of occlusal wear the periodontium maintains its integrity as observed in the Eskimos is discounting a great deal other factors which are contributory to a healthy periodontium. Racial tendencies, general health of the individual, nutritional aspects of a systemic nature, etc., are some of these factors. It is just as easy to show groups of patients without occlusal wear whose peridontium is equally intact. The fact that in the first three degrees of occlusal wear Williams determined that there were no facial changes would indicate to me that active compensating eruption did occur and this is corroborated in microscopic material of humans. His measurements on the skull were of course static at the time and even though they were ethnologically of a similar group he had no means of comparing his measurements with earlier and later aspects of the same specimen. That in the fourth degree of wear the face did close is probably explained by the fact that the wear took place to a greater extent than the active

eruption was able to compensate. One must consider terms used carefully. Eruption should mean the eruption of a tooth from one point to another with the periodontium following this eruption both at the alveolar crest and at the fundus.

One might speak more intelligently of eruption as elevation of the tooth if it projects itself occlusally from the socket without the periodontium following and therefore producing a clinical recession. It is very difficult sometimes to determine whether this is what occurs or the gingiva has receded. Passive eruption is that process which is exemplified in the proliferation of the epithelial attachment in an apical direction, and the height of the attachment following in the same direction. This can also be interpreted sometimes clinically as recession.

THE BLOOD SUPPLY OF THE GINGIVAL TISSUES

by WALTER COHN, D.D.S., *Philadelphia, Penn.*

Dr. Cohn used a very interesting technique of profusion with India ink on the blood supply of the gingiva of dogs. His slides were beautifully done and showed the amazing vascularity that the gingiva has. He showed the arterioles of the gingiva proceeding supraperiosteally along the labial and buccal aspects and the lingual and ending in an anastomosis over the crest of the alveolar process in the gingiva itself. These were connected with the arterioles which could be plainly seen coming up through the supporting bone in the interseptal alveolar bone and emerging at the crest. Some of these arterioles do of course anastomose with the vessels in the periodontal ligament but the main supply of the periodontal ligament is shown to be from the interdental arteries by means of penetrating arterioles into the periodontal ligament. Two conclusions can be drawn from this very interesting and excellent material, one that occlusal trauma or force would have very little opportunity of injuring the vascularity of the gingiva since the supply is away from and independent of any result of the force. Two, that since much of the gingiva is supplied by supraperiosteal arterioles the health of the gingiva can be improved by massage and massage can affect the speed and degree of vascularity.

This paper was discussed by *Dr. Thomas* of the University of Washington who agreed with the findings in general and pointed out that lymph vessels were not shown in this manner and since they are afferent might be affected by occlusal force in the periodontal ligament. Of course the lymph system is similar to the vascular system and the lymph vessels would follow a similar course supraperiosteally as well as intra alveolar.

THE REATTACHMENT OF THE HUMAN PERIODONTAL TISSUE FOLLOWING SURGICAL DETACHMENT

by MELVIN L. MORRIS, B.S., D.D.S., *New York, N. Y.*

Dr. Morris's paper was a type of study that has been done before and he pointed out that of course he was dealing with repair and that repair when it occurs can be somewhat the same whether or not it is connected with the gingival crevice. This is a problem we have been wrestling with for a long time and must be properly evaluated. That we do get reattachment sometimes in a pocket, with its accompanying pathology, has been established. This is not to be compared with repair which occurs as the result of surgical injury or destruction. Dr. Morris reported that he used for his study anterior teeth of humans that were to be sacrificed for prosthetic reasons. He operated on the labial aspects of centrals and created a surgical detachment by means of a bur and attempted some curettage to remove the adhering particles of periodontal ligament and cementum. He made definite notches with a round wheel at the bottom of his detachment and at the crest or the margin of the gingiva before detachment. He

found that he was able to show reattachment of the detached tissues in 10 of the 11 cases on which he worked. He found also, in substantiation of other similar studies, that in the reattachment of connective tissue the fibers are usually parallel with the long axis of the tooth instead of at right angles as we usually see them in the undisturbed periodontal ligament. New cementum is deposited and a complete reattachment is obtained. He did find however that accompanying surgical detachment of this nature that a certain amount of the gingiva is lost and it never reattaches to the original height of the margin.

COMMENTS

All of this is to be expected because the conditions of surgical injury are not entirely similar to those that are present in a pathologic pocket. All oral surgeons who use flap operations in various kinds or surgical techniques know and realize that this repair does obtain and is a clinical expectancy. This kind of detachment and the very narrowness of the detachment are conducive to the formation of a good blood clot from the surrounding healthy bone and connective tissue. It must be remembered that in surgical detachment the epithelium has had no opportunity to proliferate apically and therefore there is no barrier to the ordinary processes of repair.

The paper was discussed by *Sigurd Ramjford* who reiterated that this type of healing is like all surgical detachment as mentioned above and as such is to be accepted as repair. He showed some of his own sections on monkey material which showed repair of areas which he had destroyed *below* the alveolar crest where by means of curettage he had removed all of the cementum. This again is to be expected and not comparable to the pathologic pocket where the bottom of the pocket is always above the alveolar crest on the side affected. Very obviously if one curettes below the alveolar crest or rather apically of the alveolar crest one is then dealing with healthy tissue which has every opportunity of repair.

ALVEOLAR BONE CHANGES FOLLOWING THE ALTERATION OF MASTICATORY FUNCTION IN THE RAT

by SIDNEY L. HOROWITZ, B.S., D.D.S., AND HARRY H. SHAPIRO, *New York, N. Y.*

This was a very enlightening work done on thirty day old rats in which the temporalis in some, the masseter in others and the anterior belly of the digastric muscles were severed and rendered non-functional. The rats were fed on the standard MacCullem diet. This is a very provocative paper and those of you who are interested should refer to it when it is published for the details. In the case of the group in which the temporalis was removed on only one side the mandibular incisors deviated toward the operated side with an accompanying reported resorption of the coronoid process on that side. In the case of the severing of the anterior belly of the digastric a class three relationship was obtained. In the case of the sectioning of the masseter muscles unilaterally the greatest changes were seen, both the maxilla and mandible were deviated and there was a disturbance of occlusal relationship with open bite. The mandible appeared to be smaller than normal and yet a typical class three relationship obtained. The occlusion was traumatically disturbed. The characteristic finding both roentgenologically and histologically was a loss of alveolar bone. This was primarily exemplified in the x-ray as osteoporosis and in the histologic finding as a lessening in the number of trabeculae and not in the destruction of bone at the alveolar crest.

In other words the picture produced that which is typical of what we know as disuse atrophy, a disturbance of the masticatory function, disturbed functioning of the teeth in their occlusal relationship and the result was the loss of supporting bone. This parallels findings of Jones and Simonton in an earlier similar investigation. Dr. Horowitz who read the paper emphasized that no pocket formation was found in any

of the animals and that there was no inflammatory reaction. The finding was typical of disuse alveolar atrophy.

Dr. Joseph Weinmann discussed this paper and made some very interesting observations which were, in my opinion, a good commentary on the importance of interpreting results. He complimented the authors of the paper on their ability to dissect the muscles and sever their connections in thirty day old rats which of course is a very difficult thing to do. In the instance of the reported resorption of the coronoid process his explanation was that since the temporalis was removed from its attachment on the coronoid process the coronoid stopped its pattern of growth because of the lack of the functional stimulus, and that the condylar process of the mandible therefore continued to grow in its usual manner and that the finding then appeared as though the coronoid process had resorbed which in fact was not the case. In the interesting finding, after the removal of the anterior belly of the digastric of a shorter mandible with a class three relationsip he felt this could be explained on the basis that the maxilla of course continued to grow with the accompanying distal shift of the molars which is typical of the rat; while the mandible having been deprived of its functional stimulus to growth stopped growing and the molars stopped their distal shift and consequently the rather unique result of a class three relationship with a smaller than normal mandible.

Dr. Weinmann pointed out that the finding, illustrated in the histologic sections of a reduced density of the periodontal ligament, in these animals, was of course the replacement of a dense fibrous ligament stimulated by function to that of a retentive membrane or more properly a periodontal membrane. I think that the use of these two terms, the ordinary healthy connective tissue attached to the alveolar process at the cementum of the tooth should be known as a periodontal ligament while a non functioning attachment more properly a periodontal membrane.

THE EFFECT OF TRAUMA TO THE PERIODONTAL MEMBRANE ON THE SPREAD OF GINGIVAL INFLAMMATION

by JOSEPH WEINMANN, *Chicago, Ill.*

This was a report of work done on rats and attempting to answer the question of how does inflammation progress and how can it contribute to the destruction of the periodontal membrane as a result of traumatic injury. The method of the experiment was to place a piece of rubber dam in the interseptal space between two molar teeth of the rat and force it in quite definitely. As a result the two teeth were spread apart showing the typical effect of trauma and at the same time the interseptal gingival papilla was irritated and inflammation ensued. The effect as one can see is not only exerted upon the two teeth in which the rubber dam was placed but also those adjacent. The characteristic findings of change in tooth position, as a result of tooth movement or trauma, were ascertained—that of alveolar resorption on the pressure side and bone apposition on the tension side. But an interesting new observation was made and that is that on the tension side, and only on the tension side, and in the area where the rubber dam irritated the interdental papilla the inflammation progressed not down through the alveolar bone as one would expect but through and into the periodontal membrane. This occurred only in the space where the irritation had been produced.

One might draw a conclusion from this experiment, needing confirmation, that traumatic injury may accentuate inflammation already present. Dr. Weinmann made some very interesting comments about the structure of the periodontal ligament as seen particularly on the tension side. It has always been believed and assumed that the periodontal fibers run directly from the bone to the tooth and conversely. This of course has never been actually observed but has always been assumed. Weinmann feels that upon careful analysis one can detect in the middle of the periodontal ligament the intermediate layer one sees so definitely when the tooth is erupting. The fibers of the periodontal ligament actually do not travel directly and continuously from the alveolar bone to the cementum of the tooth but rather they join at this intermediate layer in an

interwoven sort of mechanism and that when traumatic injury occurs these fibers are not torn but they unravel. This might very well account for a path, and this is my own idea, which inflammation might be able to follow. Another interesting finding in this study exemplifying the terrific ability for repair to take place, was that on the tension side it was noted that there was a terrific increased mitotic activity. This of course was not noted on the pressure side since the vascularity would be impeded and alveolar resorption of the under-mining type would begin to take place very soon.

TREATMENT PLANNING OF DEEP OVER-BITE CASES

by EVERETT J. MARGOLIS, *Boston, Mass.*

This was a comprehensive paper and emphasizes again the need for a more correlated teaching between Orthodontics and Periodontics. Dr. Margolis restricted his comments to the overbite and said definitely that the overbite alone was not an entity but that it accompanied various and many different kinds of malocclusion. He urged the use of the Cephalogram in analyzing the effect and importance of overbite and its relationship to periodontal disease. I think this is an avenue of teaching and study that we might well pursue and enhance our knowledge of the value of this important measuring device. Dr. Margolis said that in modern man the overbite was often due to the crowding of the lower anteriors and the impaction of third molars, which were two clinical symptoms which had to be taken together as they both affect the problem. In considering the variety of etiologic factors in the development of the overbite he referred his audience to his article in the June issue of the American Journal of Orthodontics and I think that those who are interested might better pursue this article rather than have me attempt to report his concepts. He pointed out that the proportion of overbite was often dependent upon the procumbency of the upper anterior teeth. He also pointed out that whether or not it was a periodontal problem had to be determined by the Periodontist and that it was perfectly possible to have an overbite without a periodontal problem. Many overbites are better off left alone and that no ensuing tragedy will result therefrom, that sometimes the correction of the overbite was worse than the original malformation. He pointed out the practicality of the use of the Hawley appliance and often by its use an overbite can be corrected with accompanying incisal correction by grinding. Several precautions should to be followed in the use of the Hawley appliance and one was that no greater opening than one millimeter should be used in separating the molars. He believes that such a Hawley, in order to increase vertical dimension and therefore help to correct the overbite, should be worn all the time including the periods of mastication. The maxillary teeth are capable of extrusion along with their periodontium and therefore coming into contact at an earlier time and then the Hawley can be remade opening it again another millimeter—never more.

In general terms these appliances are usually worn at least a year and sometimes after their removal the teeth become subsequently depressed so that the case is back to status quo. He feels that the whole treatment on this basis is the allaying of a symptom by means of a crutch. Again he emphasized that the use of the Hawley appliance should be carefully diagnosed and in many instances, where properly indicated, could be a successful appliance. In cases of overbite, accompanied by crowding or buckling of the lower anteriors, he suggested the removal of one of the teeth in order to bring the remaining teeth forward in a better relationship to the lingual of the upper anteriors. This has to be done too with proper diagnosis and consideration and he feels that the less residual space remaining after extraction of one tooth the more practical is the treatment. He feels very definitely that these teeth when moved into proper align-

ment should be moved bodily by appliances that can accomplish this and not just tipped forward or mesially or distally.

The paper was discussed by *Dr. Louis Fox* of the Columbia Dental School. Dr. Fox is an expert in reconstruction and approached this problem from the standpoint of reconstruction. He reiterated Dr. Margolis' idea that the overbite "never walks alone" and that it is dependent a good deal upon the axial inclination of the incisors. The proportion of the lingual surface of the upper teeth and its relationship to the incisal edge of the lower teeth is important in determining what therapy is to be used. He pointed out that Dr. Margolis did not mention overjet and that he feels that overbite and overjet accompany each other and in some instances, as far as periodontal disease is concerned overjet compensates for any problems that overbite might precipitate. He also feels that Dr. Margolis' concept of overbite not causing serious periodontal problems was redundant and that it does more often than we realize. Dr. Fox emphasized the consideration of the patient's free-way space in any corrective therapy in overbite and of course that the free-way space should never be exceeded. He feels that this whole problem must be evaluated on a mass basis and points out that Marshall Day, at Tufts College, is now conducting a statistical evaluation of malocclusion and ill health, to which he refers us. To repeat, periodontists do not know enough concerning the relationship of malocclusion to periodontal disease and that it behooves us to be more cognizant of the principles being pursued in orthodontics.

PROTEIN DEFICIENCIES IN ANIMALS AND EFFECT ON THE ATTACHMENT APPARATUS

by HENRY M. GOLDMAN, D.M.D., *Boston, Mass.*

This was an extemporaneous presentation and was reported only in part and in particular reference to the work done on the Spider Monkey which Goldman has been doing for some little time. This project is a continuing one and more reports will be forthcoming later. Appropos of this material I would like to refer you to the article Drs. Frandsen and Becks on a similar study. This latter article is published in the Journal of Periodontology, July 1953. Goldman fed his animals a diet containing no protein but with Vitamin supplementation. He found that there was a definite lesion produced thereby which affected the interdental papilla and the alveolar bone. This is a very interesting finding since it points toward more knowledge of the periodontal disease we speak of as periodontosis. Periodontosis seems to be essentially a degenerative process occurring in the absence of inflammation but further complicated by it. Goldman found that the lesions in his monkeys were produced in the young and therefore there seems to be an age factor which ties up with the clinical picture of periodontosis. I should like to point out that this finding of Goldman's is not identical with that which we see in the human and call periodontosis, but there are interesting similarities. Here is a nutritional deficiency which according to Goldman has selectivity for the jaws and this in itself is of course important. Typical of the lesions so produced is the degeneration of collagen in the periodontal ligament and also in the gingiva and there are fewer cells. In some instances resorption of bone surrounding the entire root and with a border of what appears roentgenologically to be an increased density of the cortical bone. The characteristic feature of osteroporosis in the supporting bone is recognized. The fact that there was no histological confirmation of the increased density seen in the radiograph is understood by the fact that in the microscopic section one only sees a small segment of the bone and is unable to detect the degree of combined calcification. Such a finding can only be visual in the x-ray where the confluences of the cortical plate or cribiform plates are evident. Since it was a preliminary report more information concerning this study will be available when the paper is published and again in similar papers written by others.

DISCUSSION

Dr. Paul O. Boyle of the University of Pennsylvania discussed the paper. He confirmed the finding of the widening of the periodontal membrane and the loss of alveolar bone in animals fed a diet of 0 protein. Dr. Boyle pointed out that Vitamin C deficiency diets produce somewhat the same condition. Here of course Vitamin C is important in the production of collagen and in differentiation of fiberblasts. Dr. Boyle injected the problem of sex into this problem which in his experience has complicated the picture. He pointed out also that the complexity of protein metabolism can be varied by extrinsic factors as well as intrinsic factors and also spoke of the difficulty of correlating radiographic density and histologic findings.

SYMPOSIUM ON ORAL LESIONS

Dr. David Weisberger, D.M.D., M.D., of Harvard presented the clinician's viewpoint and chose for his subject *Lymphoma.* This and the one following it by Dr. Gorlin, who covered the histopathology of Lymphoma, were classical presentations and of scholarly interest. Dr. Weisberger probably presented it before our group on the basis of the fact that dentists should be alert to enlargement of lymph nodes which is a diagnostic signal for many different diseases. Dentists can alert the patient before a particular disease has made itself more systemically manifest. He pointed out that swollen lymph nodes are found in Leukemia, in the Lymphomas, in Gauche's disease, in Sarcoma and Hodgkin's Disease. He also emphasized that a chronic enlargement of a lymph node is rarely due to inflammation. This means of course that when we find these conditions in our patients we should refer them for more specific diagnosis. The Lymphomas are in the main malignant diseases divided into Lympho-Sarcoma, which is primarily invasive from the lymphoid tissue, the Reticular cell Lymphoma which is also invasive but has a different type of cell more like the reticular cells of the reticular system and Hodgkin's disease, which is also a disease of the lymph nodes primarily and characterized by the Dorothy Reid Sternberg cell. Hodgkin's disease has of course several types—the Granuloma which is characterized by fibrosis and necrosis and which the Dorothy Reid Sternberg cell is diagnostic. The Sarcoma which is composed of large cells with no fibrosis and no necrosis is again positively diagnosed by the Dorothy Reid Sternberg cell. The giant follicular type which is characterized by hyperplasia of the germinal centers of the lymph nodes and a confluence of the entire node. The etiology of these diseases is of course unknown. Some feel that it has been associated with Tuberculosis, some that it is a very definite neoplasm, others that it is initiated by virus infection.

Dr. Weisberger spoke concerning the pathology of Hodgkin's disease pointing out that the lymph nodes involved were very often the cervical and then the axillary and finally the inguinal, in that order. In about 25% of the cases bone is involved which is manifested by destruction. One of the first signs that may be seen by a diagnostician is a painless lump, often in the neck. Accompanying this there is an intermittent fever and a body itching. All these are symptoms of Hodgkin's disease. The itching is often accompanied by bronzed skin and peculiar papular skin lesions. The fever is irregular and follows what is called a Pal Epstein change.

Dr. Gorlin from the University of Iowa, and now with the Navy, then gave a very scholarly dissertation on the histopathologly of these conditions particularly in reference to the changes seen in lymph nodes and the lymphocytes concerned. This was eloquently portrayed and does not bear repeating here since it was a comprehensive study meant for those who are particularly interested.

COMMENTS

This whole problem, even back to the consideration of the origin of the lymphocytes, be they from a stem cell or from an undifferentiated mesenchymal cell, is one that has many gaps in its knowledge but

one that pathologists are working with all of the time. As you probably know there is no particular specific treatment for a Lymphoma. Nitrogen mustard has been tried with varying success as has also radio active phosphorus, radiation in some instances is beneficial. Hodgkin's disease is often very slow in its development and patients live a long time before the terminal effects set in and then on the other hand it may go very rapidly. There are some instances where lesions connected with Hodgkin's disease are visible in the oral cavity. They are often abraded lesions similar to apthous ulcers but of a different texture. They are never diagnostic however but certainly when seen, in connection with chronic enlargement of lymph nodes, steps should be taken toward a proper diagnosis. The diagnosis is usually made by the removal of a lymph node and the examination histopathologically for the Dorothy Reid Sternberg cell.

SYMPOSIUM ON BONE AND BONE RESORPTION—I BIOCHEMICAL AND BIOMECHANICAL ASPECTS OF BONE RESORPTION

by FRANKLIN C. MCLEAN, M.D., PH.D., *Chicago, Ill.*

Dr. McLean's paper was a classical and scholarly presentation indeed. It was well written and well presented and summed up what we know about bone resorption today. He said that our knowledge of bone resorption had not changed very much since Koelliker's classic book of eighty years ago when he first described the function of the osteoclast in this process. Dr. McLean pointed out that bone is composed of a matrix, mineral and serological content which is all in solution of a very complicated structure. He also pointed out that there are gaps in our present day knowledge of the process of bone resorption but there are some features that now have a preponderance of evidence. In order to resorb bone the matrix, the mineral and the serologic content must be reduced to a compound or product which is soluble in water and can be transferred to the blood. The interstitial tissue of bone, between the osteocytes that is, is composed of water, organic material and inorganic salts which make up about 65% of its content. The organic material is fibrous or collagen and the ground substance is a glyco-protein which is thought, at present, to be chondroiton sulfate.

McLean feels now that the osteoclast is concerned with the resorption of all of these materials at the same time and that it probably acts as an enzyme producer which changes these materials into a soluble state. The feeling that the osteoclast may act as a phagocyte of these materials is open to a considerable amount of question, specimens that he has seen will show granules of calcific salts around and close to the osteoclast but not necessarily within it. That the osteoclast engulfs the osteocytes has been shown but of what significance this may be is also open to question. The origin of the osteoclast may very likely be from a group of osteoblasts combining together to make a multi-nucleated cell which acts much as a giant cell anywhere in the body. Foreign body giant cells are specific in the removal of foreign materials and are active in many conditions we see in pathology. In the resorption process we have considered the necessity to resorb the mineral of bone first of all. Formerly this was felt only to be possible in the presence of acid of sufficient PH to produce dissolution. There is now the very good possibility that the osteoclast aids in forming chelating agents which are compounds characterized by a high affinity for calcium salts and it is perfectly possible and a logical assumption that these chelating agents are able to extract the calcium salt at the time that the matrix is being disintegrated and thereby account for the removal of inorganic material. This whole process is called Chelation (Crab like) and does not have to take place in an acid PH, as a matter of fact bones and teeth can be declacified in these agents even in an alkaline medium. Collagen can be rendered soluble by certain digesting enzymes, pepsin is one although, interestingly enough, trypsin is not. We now know there exists an enzyme called collagenase which is able to render collagen soluble.

The ground substance is rendered soluble probably by a change in polymerization or by becoming depolymerized. Gersh and others working with connective tissue ground substance have shown this to be the case and when it becomes depolymerized to a low or less complicated molecular arrangement it becomes soluble.

It is McLean's opinion that the various elements which compose the interstitial tissue of bone all become resorbed together. The osteoclast is very likely the instigator in this process and produces either the enzymes which are initially effective or the precursors for this complicated bio-chemical process. This is evidenced by the fact that they are always present at the sites of bone resorption and occupy bays of erosion in the bone. This of course is readily visible in microscopic material. As repeated before they are probably produced by a fusion of osteoblasts or are the results of a common ancestry, namely the undifferentiated mesencymal cell. The idea that resorption of bone could only take place in the presence of acids has been heretofore a difficult stumbling block but now with a greater knowledge of chelating agents and Chelation this is no longer so. He repeats that the constant appearance of osteoclasts in the process of resorption is more than incidental. He does pose the question however that if they are concerned, and he believes they are, what is the process of control, how are osteoclasts called upon to perform the function of resorption and start the biochemistry in this direction? This, Dr. McLean points out is one of the things that we have very little knowledge about and the mechanism remains for further research. He points out that even under hormonal control there are differences in reaction of osteoclasts in resorption. The use of Parathyroid extract produces hyper-parathyroidism which produces bone resorption accompanied by osteoclasts and the cell transformation can be noted.

Dr. McLean is famous for studies made on birds in this regard, particularly the pigeon during the egg-laying cycle. Study was conducted by the injection of estrogen which produces an inner structure of cancellous bone in the long bones upon which the bird could draw for the reserve supply of calcium necessary to form the shell of the egg. This process is accomplished by the osteoclast working on this inner structure of cancellous bone within the long bones of the bird but interestingly enough the integral part of the skeleton is not attacked. These are some of questions that McLean points out are still waiting explanation. The serum calcium in these birds is of course highly elevated during this period plus a phospho protein. The excess calcium in the plasma is poorly ionized and it is accompanied by a compound which has chelating ability.

In considering the growth of the skeleton McLean points out that growth of bone cannot take place without reconstruction and this reconstruction is always accompanied by osteoclastic activity. Resorption of bone is a physiologic process as well as a pathologic one and therefore there must be an organized enzymatic mechanism for it to occur. The entire skeleton and organs of bone are structurally adapted to function and this adaptation cannot take place without resorption. Bone is known to resorb under pressure, where again the osteoclast is active. Just how this takes place is still a mystery but it has been suggested that the osteoblast produces a change and liberates a product similar to histamine which induces the ground substance of the bone to elaborate another material which sends the osteoclast into action as the resorbing agent. This of course is purely a hypothesis. Dr. McLean pointed out that enamel and dentin are exempt from osteoclastic resorption even in hyperparathyroidism.

In summary Dr. McLean stated that resorption of bone occurs mainly by osteoclastic activity and this is true in all animals under parathyroid extract injection and in birds in estrogenic injection. The connection between the osteoclast and resorption is found in both normal and pathologic bone resorption—that evidently they simultaneously provide enzymes which make the interstitial substance of bone soluble concurrently with the calcium; that the newest knowledge and greatest advance in the picture is the theory of Chelation which has to be explored further.

DISCUSSION

This paper was discussed by *Dr. Balint Orban* who reiterated complete agreement with Dr. McLean in the process of bone resorption and pointed out that the conditions of alveolar resorption are of course identical, that in normal physiology apposition and resorption are due to function and that the physiological mesial drift of the teeth that can be seen microscopically, with resorption on the mesial and apposition on the distal, is an example of the same process. That in the alveolar bone there is internal reconstruction going on all of the time, no one really knows the life span of a cell and the life span of an osteocyte is not necessarily any greater or lesser than the other cells and thus bone reconstruction has to be continuous to keep the bone vital. How inflammation produces or stimulates resorption of bone is still a matter of question, it probably comes about through toxins producing tissue necrosis which in some manner directly stimulate the osteoclasts, or it is possible that inflammation produces pressure, which can be ascertained in our field, stimulates the osteoclast to resorb bone in that manner. In trauma the compression of the connective tissue can produce pressure on bone and therefore resorption and on the tension side the functional stimulus produces apposition. In degenerative diseases such as periodontosis the resorption can be stimulated by some intrinsic factor, mainly the depolymerization of the connective tissue, and can elicit certain enzymes from the ground substance of the bone or affect the ground substance of the bone at the same time and thereby stimulate osteoclastic function.

Dr. Joseph Weinmann stated that he was in complete accord with McLean's paper and again the osteoclast must be concerned with resorption because of its close histologic association. He pointed out that rats which were studied in rickets during the growth period showed no resorption in the areas of bone reconstruction because the resorption was unable to take place but that huge numbers of osteoclast were always seen in this area waiting to go to work. (The fact that in rickets osteoid tissue is not calcified probably inhibits a resorption process.) Weinmann did not agree with McLean that osteoclasts are formed from a fusion of osteoblasts but more particularly from reticular cells or undifferentiated mesenchymal cells. If an implant of boiled bone is made it is resorbed and completely surrounded by osteoclasts. He pointed out too that the internal resorption of dentin by osteoclasts (odontoclasts) does take place. He also said that pressure alone producing resorption was an over-simplification of the entire process and that in order for resorption to occur a change in the intercellular substance must be produced.

COMMENTS

In the light of the preponderance of evidence in the direction of the osteoclast's concern, in the resorption of bone and that bone is resorbed in its entirety, the ground substance the serum fluid and the mineral salts all at the same time and that with the theory of Chelation a possibility in explaining the solution of inorganic material, I think it is hard to conceive that resorption of bone takes place by a de-mineralization of only the inorganic salts leaving the matrix intact. It is further difficult to embrace that this decalcified matrix can be the same type of substance known as osteoid which is newly laid down by osteoblasts and can receive calcium on the basis of re-calcification.

SYMPOSIUM ON UNDERGRADUATE AND GRADUATE TRAINING IN PERIODONTIA RELATED TO CERTIFICATION

There were four people who took part—Dr. Thomas from Washington, Dr. Henry Goldman from Boston, Dr. Harold Leonard from New York and Dr. James Blayney from the University of Chicago, representing the Council on Dental Education.

This symposium was not too satisfactory. The speakers covered the subject but the whole problem of undergraduate and graduate training in Periodontology is now undergoing critical review and the criteria for certification is subject to many things. The precepts and requirements laid down by the Council on Dental Education, the ability of schools to offer the proper courses leading to certification and an intelligent analysis of the need and experience and training required in order to produce an individual expert to a more than ordinary degree in the treatment of Periodontal diseases.

To resolve this problem the Academy has now a standing committee on education which is giving exhaustive study to the overall picture and the section of Periodontia of the American Association of Dental Schools will devote its efforts and meetings to a review of this entire problem along with a study of Methodology in the teaching of both undergraduate and graduate periodontics.

PANEL DISCUSSION ON TREATMENT

On Saturday morning, at the closing part of the scientific meeting, a panel on the discussion of the various surgical treatments of marginal gingivitis and periodontitis was conducted. *Dr. Bernard S. Chaikin* of Boston spoke on curettage. He gave a very interesting description of subgingival curettage and pointed out that for successful accomplishment the crevicular epithelium, including the epithelial attachment, must be removed in the process. He also stated that pockets can be reduced by shrinkage or by approximation to the tooth or by regeneration (reattachment). That if the inflammatory process continued, pathology of the pocket was still in existence, then gingivectomy should be resorted to. These papers were only of ten minutes' duration each so that no attempt was made to elaborate or be extensive in depicting techniques. The instruments that Chaikin uses are primarily curettes and he points out the difficulty and skill which is necessary to do complete subgingival curettage and accomplish that which is biologically necessary in the process.

Dr. Robert Gottsegen of Columbia University, New York, discussed Gingivoplasty and talked about the use of these procedures in the reshaping of the gingiva, in order to correct areas receiving undue irritation because of their abnormal contour and also a matter of prevention. He showed some very interesting slides of results obtained in this procedure. Dr. Gottsegen had a very interesting operation for closely attached labial frenums which was interesting to see. He resects the mucobuccal fold below the frenum and therefore detaches it and applies a plastic splint over the teeth and down into the mucobuccal fold in order to control healing. Some of his results were very interesting indeed, in these cases where the frenum is very closely attached contributing to the extension and formation of labial pockets particularly on the lower anterior teeth.

Dr. Richard Chase from Orlando, Florida, spoke on gingivectomy. This was a very good recapitulation of ordinary gingivectomy in which the incision was made at the depth of the bottom of the pocket and the granulation tissue removed and the root surfaces curetted and scraped decisively. Chase's contribution mainly was emphasis on the curettement of the root surfaces which is a logical and necessary part of any gingivectomy procedure.

Dr. Schluger, of Columbia talked on osteo resection or osteoplasty. He talked at great length about form and function. Essentially he covered various techniques under various conditions in which bone is removed on an architectural basis. Obviously there are many cases in periodontal disease where bone can justifiably be removed in order to produce an architectural condition which will respond properly to function. This is particularly true

in intrabony pockets, in tortuous involvements on the lingual of an intrabony nature, in bifurcation instances, etc. Dr. Schluger pointed out that this bone is removed for purely architectural reasons and not because it is necrotic. On this basis the technique has validity.

Dr. Jack Bolin from South Norwalk, Conn., talked on the use of the flap approach in gingivectomy. He pointed out that this is a very helpful adjunct and very successful when indicated, and not to be looked upon as heroic since flap approaches are common techniques in all oral surgical procedure. He said it is indicated when gingivectomy per se has been unsuccessful or in combination with gingivectomy and subgingival curettage. It is particularly indicated in intrabony pockets, in bifurcation and tortuous procedures. Obviously his presentation and Schlugers went hand in hand because after reflecting the flap the architecturai reconstruction of bone can be proceeded with, with greater accuracy. He named as an advantage greater access and that this could also result in more conservative treatment because the entire problem is visible. The analysis and understanding of the particular pathologic situation is simplifitd and leads towards greater success. He pointed out that it is very hard and difficult to predict success in this technique, as it is in most therapeutic treatments for periodontal disease.

Letter to the Editor

Dear Editor:

In reference to our article entitled "Results of a Three Year Study of Vincent's Infection at the Great Lakes Naval Dental Department" appearing on pages 187-194 in the 24 vol. of the July, 1953 issue of the Journal of Periodontology, a mistake has been brought to our attention on the three tables of monthly attack rates. An errata is proposed to show the corrected values:

"In Table A the mean and median for Vincent's case should be 1.35 and 1.34 respectively. The mean and median for unclassified gingivitis should read 1.88 and 2.02. In Table B the means for Vincent's and unclassified gingivitis are correct; however, the median for Vincent's should read 1.44 and for unclassified gingivitis—2.48.

The mean and median for Vincent's in Table C should read 1.60 and 1.56 respectively, and the mean and median for unclassified gingivitis should be 6.41 and 5.93.

The reason for this confusion is not fully understood since it is apparent that the means of Table A and C are reversed, but the medians continue to be a mystery. I hope you will find it proper that these corrections are included in an early issue of the Journal of Periodontology.

With kindest regards,

Wm. J. Carter, D.D.S., M.S.

REPORT OF THE 1952-53 COMMITTEE ON RESEARCH OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

Your Committee believes that one of the important functions that the Academy can perform is that of a correlating body, a central clearing house of Periodontal Research being prosecuted by our teaching institutions. To this end your Committee has compiled a composite picture of research projects now being pursued in these institutions. Copies of the compilation are here available for those who are particularly interested.

The projects submitted were arbitrarily divided into three groups namely, Biologic (A), Bio-Clinical (B) and Clinical (C). The purpose of this division was to gain some idea of the direction and motivation that our research is taking today. Research in our field, as in many fields, must eventually find a clinical application. Clinical research should be of such calibre and direction that it points the way or avenue to be explored by those concerned with the Biologic aspects of our problems. The Bio-Clinical projects being conducted should be the connecting link between the pure clinical application and the basic biologic concepts.

Our study reveals that there is insufficient correlation between the clinical projects and the biologic. While all of the studies being conducted have a very definite relation to problems in Periodontics, the overall picture does not present an orderly continuity. The clinical group of studies now being conducted can be most pointedly criticized. This is a heterogeneous group of projects having no particular direction. They all pertain to our problem, of course, but have been dreamed up individually,. stimulated by a variety of interests. One can see clearly the enthusiastic influence of drug houses in evaluating their products, often resulting in needless repetition. There are too the hobby horses ridden by enthusiasts for certain principles already evaluated.

This criticism is not pointed at the fact that any of these projects are redundant, but rather to the salient point that there is a lack of correlation in either a general continuity or between the institutions conducting research.

Our recommendations for the future are that the Committee on Research in Periodontology of the Academy be an active working committee whose function it should be to assist in an overall correlation and projection of research projects in our field. It is further recommended that a composite study, such as the present one, be made by this committee each year, and that it be published in a particular issue of our Journal. This would serve as a catalog of Research in Progress and act as a reference guide for everyone interested in our overall problem. If in many instances it could be enhanced with abstracts, that would only add to its general value. We can visualize that this committee from these studies could organize all inclusive programs, aspects of which could be prosecuted by various institutions with the net results contributing to a unified whole of importance to the practice of Periodontia. In performing such a function this Academy would be additionally furthering its objective and serving the profession of Dentistry.

Compilation of Periodontal Research in Process in American Dental Schools

University of Alabama

(A) Bacterial & Immunological aspects of streptococcus salivarius
Mucopolysaccharides in saliva

(B) Relationship of Vaccinia Antibody levels in aphthous stomatitis
Neuroanatomical study of oral structures using electrographic technic

(C) Influence of orange juice on teeth, oral flora & Perio. (HAMSTERS & HUMANS)

Baltimore College
(A) Dynamics of pocket formation (from several angles)

Baylor University
(A) Relationship of alveolar resorption to denture prognosis after extraction of periodontally involved teeth
(C) Terramycin mouth wash in apthae
Hydrocortone ointment in gingivitis

University of California
(A) Effect of controlled radiation on periodontium (dogs)
Histopathology of gingiva in dilantin sodium
Histo-chemical study of effects of hydrocortone oint.
Relation of ovarian function to Perio. (monkeys)
Interrelation of endocrine disturbance to Perio. health (human)
(B) Pantothenic & nicotinic acid levels in Perio. patients
(C) Emotional disturbances in Perio. disease
Effects of electrocoagulation applied to Perio. on dental pulp

Columbia University
(B) Four studies on reattachment (animal & human)
(C) Stress differences on Perio. between fixed and removable appliances

Georgetown University
(A) Blood picture in Periodontia
pH of periodontal pocket & glycogen content of tissues
(B) Nutrition in Periodontia
Topical anesthesia in Periodontia
(C) Relation of root volume to length
Toothbrush in gingivitis

Harvard University
(A) Metabolism of oral mucosa
Physiology of alveolar bone
(B) Virus diseases affecting the mucous membrane

University of Illinois
(A) Enzymology & Morphology of salivary glands of rats
(B) Chemical inhibition of calculus formation
(C) P.M.A. method of assessing gingival disease

Indiana University
(A) Histopathology of periodontal pocket
Dilantin sodium on rat gingiva
(B) Wound healing in electro surgery
(C) Use of Tannic acid in surgical dressing

University of Iowa
(A) Altered capillary permeability as a contributing etiological factor in periodontal disease

University of Kansas City
(A) Role of collagenase in the destruction of periodontal fibers
(B) Histological tissue changes produced by purified enzymes

University of Louisville
(A) Causes of Oral inflammation using filtrates of cultures of material from unclean mouths

Loyola University—Chicago
(A) Epithelial attachment
Saliva in calculus free & present patients
(B) Nature of Materia Alba
Effect of "Prophie"
(C) Airbrasive on gingiva
Occlusal disharmonies in students

Marquette University

(A) Parasites in the mouth related to various stages, severity and forms of ulcero-membranous necrotizing gingivitis

University of Michigan

(A) Status of the epithelial attachment

(B) Conditions in periodontal pocket after scaling

University of Minnesota

(A) Mitotic activity of periodontium of rat molars (physiologic & pathologic)

(B) Histologic proof of reattachment
Effect of controlled horizontal forces on Periodontium (Monkey)

(C) Studies in tooth mobility

New York University

(A) Effect of STH & insulin on growth of the periodontal structures of the rat
Blood & urine studies in Perio.

(B) Effects of Vitamin therapy upon hormonal gingivitides

(C) Effect of Cortisone on clinically inflamed tissues

University of North Carolina

(A) Sex dimorphism in the mouse based on changes in salivary glands

(B) Relationship of salivary calculus to other stone formation

(C) Effect of colloidal selenium on the periodontal tissues

Northwestern University

(A) Variable occlusal stresses on periodontium of monkeys
Vitamin therapy in gingivitis

(B) Therapeutic value of Heliogen in gingivitis

(C) Use of Tryptar in chemical debridement

University of Oregon

(A) Mitchell index of gingival changes in alloxan-diabetic hamsters
Morphology of endamoeba gingivalis

(B) Histo-morphology of Maxillary-lingual retromolar triangle

(C) Mass study of gingival disease in children

University of Pennsylvania

(A) Blood supply of the gingiva & Periodontal membrane (dog)

(B) Therapy of & possible reattachment in suprabony pocket

(C) Study of normal gingiva (clinical & biopsy)

Temple University

(B) Tooth root anatomy, A factor in mobility

(C) Clinical normal gingival crevice
Impossibility of Zero crevice

University of Texas

(A) Oxygen uptake of the gingiva
Melanoblast in the inflamed gingiva
Electron microscopic study of the gingiva
Reticulo-endothelial system of the gingiva
Connective tissue fibers of the marginal gingiva

(B) Clinical & bacteriological study of necrotic gingivitis
Morphological studies of a filamentous micro-organism from tooth debris

(C) Effect of periodontal treatment on C.T. fibers of marginal gingiva

Medical College of Virginia

(B) Bacteriology & Histopathology of periodontal disease

Washington University

(A) Comparative studies of skin & mucosal changes in experimental stress

(B) Masticatory patterns & periodontal disease

(C) Hydrocortone acetate in periodontal therapy

Western Reserve University

(A) Effect of Bacterial filtrates, products or purified chemical compounds upon ground substance of tissues (cyto-Chem.) methods (Hamsters)

Tufts College

(A) The effect of Hypothyroidism upon the periodontium of the albino rat
The effect of testosterone and alpha estradiol on the periodontium and bone of young adult mice
The effect of testosterone and alpha estradiol on the periodontium and bone of adult mice receiving cortisone systemically
The effect of subcutaneous administration of chelating agents upon bone of adult mice
The role of increased functional stress in pocket formation in dogs

(B) Epidemiology of periodontal disease
Histochemical study of aging in the gingiva

(C) A series of studies on the removal of calculus by non-mechanical agents
The effect of topical applications of cortisone ointment upon specific forms of gingival disease
A clinical study of the gingival esthetics following gingivectomy.

University of Southern California

(A) Histological investigation on gingival keratinization

(B) Histological investigation on the removal of the epithelial attachment and the crevicular epithelium by instrumentaton
Clinical and histological study of the effects of tissue-reducing agents used in soft tissue pockets

(C) Clinical investigation of the effects of subgingival curettage as opposed to subgingival scaling

DIGEST

Schools reporting	28	Number of projects	100
no research	7	(A) Biologic	44
no report	7	(B) Bio-clinical	30
		(C) Clinical	26

Irving Glickman, Leonard Rosenthal, Harold Ray, Chairman.

COURSES OFFERED BY TUFTS COLLEGE DENTAL SCHOOL

The courses in Periodontology listed below will be presented at Tufts College Dental School throughout the academic year of 1953-54. Information and application may be obtained by writing the Director, Division of Graduate and Postgraduate Studies, Tufts College Dental School, 136 Harrison Ave., Boston 11, Mass.

DPG. 602—Participation Course in Periodontology—Monday through Friday, April 12-16, 1954
Tuition $150.00. Class limited to twelve. Dr. Irving Glickman and Staff

DPG. 603—Kirkland Gingivectomy Technique—Monday through Friday, May 17-21, 1954
Tuition $125.00. Class limited to twelve. Dr. Olin Kirkland

DPG. 604—Occlusal Equilibration—Each Monday for five weeks from Jan. 11 to Feb. 8, 1954
Tuition $150.00. Class limited to twelve. Dr. Edward T. Fischer

ROSE COHEN MEMORIAL LECTURE MARCH 31, 1954

The first Rose Cohen Memorial Lecture sponsored by the Division of Periodontology, Columbia University Dental School.

Essayist—Dr. Robert Moyers, Professor of Orthodontics—University of Michigan School of Dentistry

Subject—The Physiology of Centric Relation

Time and Place—Wednesday evening, March 31, 1954, at 8:00 P.M. in Amphitheatre A at Columbia University Dental School, 630 West 168th Street, New York 32, N. Y.

All members of the profession are invited.

AMERICAN ACADEMY OF DENTAL MEDICINE

The Eighth Annual Meeting of the American Academy of Dental Medicine will be held at the Penn Sheraton Hotel in Philadelphia, Pa., on Friday, Saturday and Sunday, May 14, 15 and 16, 1954. "Diagnosis and Treatment Planning" will be the theme of the scientific sessions and will include lectures and discussion groups on all phases of the subject.

A program of sightseeing and entertainment is also being arranged for those attending and their families. All members and interested dentists and physicians are cordially invited. Programs and reservations are available through the office of the National Secretary, Dr. William M. Greenhut, 124 East 84th Street, New York 28, N. Y.

THE OHIO STATE UNIVERSITY

The College of Dentistry, The Ohio State University, will present the following postgraduate courses from January 1954 to June 1954:

Anatomy of the Head and Neck, June 7-11; Clinical Oral Pathology and Diagnosis, April 26-30; Endodontics, March 8-12; Periodontics, March 1-5 and May 3-7.

Further information may be secured from Postgraduate Division, College of Dentistry, Ohio State University, Columbus 10, Ohio.

THE FIFTH ANNUAL BERKSHIRE CONFERENCE IN PERIODONTOLOGY

The Division of Graduate and Postgraduate Studies of Tufts College Dental School will conduct the Fifth Annual Berkshire Conference in Periodontology and Oral Pathology at the Cape Codder Hotel, Falmouth by the Sea, Falmouth, Massachusetts, June 20-24, 1954. The Conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields. A special feature of the Conference will be a clinico-pathological seminar with microscopes provided for all participants.

The curriculum is arranged so that mornings and evenings are devoted to professional activities with afternoons open for recreation. This is a conference for general practitioners as well as those interested in a specialized practice.

For further information and application, write to: The Fifth Berkshire Conference, Tufts College Dental School, 136 Harrison Avenue, Boston 11, Massachusetts.

UNIVERSITY OF ILLINOIS COLLEGE OF DENTISTRY

"Correction of Occlusal Disharmonies"

Thursday, Feb. 11

9:00-12:00—"Changes in the Periodontium Resulting from Trauma"
Dr. Leo Sreebny

"The Use of Plate Appliances in the Treatment of Periodontal Trauma"
Dr. Max Gratzinger

"Normal and Pathologic Changes in the Masticatory Mechanism, with Special Reference to the TM Joint"
Dr. Frank Wentz

1:30- 5:00—"Correction of Occlusal Disharmonies"
Dr. Balint Orban

(TV Demonstration and Participation by Class)

5:00- 6:00—Patient Demonstration

7:30- 9:00—Round Table Discussion

For details, write Dr. Isaac Schour, 808 S. Wood St., Chicago, Illinois.

TEMPLE UNIVERSITY POSTGRADUATE COURSES

Periodontal Pathology. This five-day course given March 1, 2, 3, 4, and 5, 1954 (9 a.m. to 5 p.m.) is designed to achieve a close coordination of clinical observation and microscopic study of pathologic deviations. Tuition $200.00.

Clinical Occlusion. This three-day course, to be given April 14, 15, and 16, 1954 (9 a.m. to 5 p.m.), is designed to present the fundamentals of occlusion in relation to the physiology and pathology of the periodontal supporting tissues. Tuition $150.00.

Applications can be made to: Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

ACADEMY OF ORAL PATHOLOGY

The Academy of Oral Pathology will hold its Annual Meeting on Sunday, February 7, 1954, in the South Ballroom of the Conrad Hilton Hotel, beginning promptly at 9:00 a.m.

The program includes the following:

"Studies of the Periodontium of Hamsters," by Dr. David Mitchell.

"Physiology of Keratinization," by Dr. S. Rothman.

"Unusual Hematomas of the Oral Regions," by Dr. J. L. Bernier.

"Some Aspects of the Histopathology of the Tongue," by Dr. George Stein.

"Spread of Inflammation," by Dr. Harry Sicher.

"Melanomas of the Gingivae," by Drs. Baldridge and Waldron.

"Symposium of Biopsy," by Dr. Kurt H. Thoma, Dr. Donald A. Kerr, Dr. H. B. G. Robinson, Dr. Paul E. Boyle, Dr. Joseph L. Bernier.

All dentists and others interested are cordially invited to participate.